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***AIR HYGIENE***

***IN***

***HOSPITALS.***

**John A. N. Emslie**

**M.D. Thesis**

**September 1906**

**University of Glasgow.**

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## SUMMARY

### **Air Hygiene in Hospitals.**

**Enslie, John A.N..**

**M.D. Thesis, University of Glasgow. (Sept. 1966)**

**59 pages of text, 39 tables, 17 figures,  
158 references.**

This thesis is in two main parts - a review and a report. In the review, the development of air hygiene as a hospital discipline is recounted before current principles and practices are discussed. The principles of airborne infection are considered under the headings of sources and dispersal, environmental transmission, and colonisation and infectivity. Similar headings are used to outline methods of control. Brief comments are included to complete the review on the types of pathogens most commonly involved in airborne infection and on the equipment and methods for use in studying them.

In the report section, details are recorded of several different investigations. A series of experiments with artificially produced aerosols demonstrated the fallacy of using a single index of measurement for naturally ventilated atmospheres, although such aerosols are useful for determining experimentally the transfer of airborne bacteria throughout a building. Also demonstrated were the inconsistencies to be found when colony-counts are performed, when unmatched slit-samplers are used together, when air-sampling is done by means



of tubing attached to slit-samplers, and when the concentration of aerial contaminants in a whole room is estimated by inference from an observed concentration at only one site in the room. A bacteriological method is described for evaluating a simple photometer with which the bactericidal light output (2537 Å wavelength) of commercial ultraviolet light tubes can be tested. Finally, details and observations are recorded of a few studies of naturally occurring aerial contamination in wards and in operating theatre suites.

It is concluded that much is known already about airborne infection in hospitals but that relatively little has been done to apply this knowledge to establish more hygienic conditions. The benefits to be derived from improved conditions cannot always be measured to everyone's satisfaction but, as a general rule, any structural or functional change which reduces either the production or the concentration of aerial contaminants should be adopted whenever possible. The acquisition of new knowledge and the refinement of that available at present is likely to be difficult because of the numerous and inter-dependent aspects of airborne infection. Probably this will require the establishment of special facilities. However, the most vital change of all which is needed is in the mental attitude to air hygiene shown by the medical, nursing and domestic staffs most immediately involved in protecting the health of patients.

# CONTENTS

	Pages
1. Introduction.	1
2. Review.	
2.1. Historical review -	
1: Before 1900	2 - 3
2: Between 1900 and 1950	3 - 6
3: Between 1950 and 1960	6 - 8
2.2. Current Principles and Practices -	
1: Airborne infection - sources and dispersal	8 - 11
2: Airborne infection - environmental transmission	11 - 13
3: Airborne infection - colonisation and infectivity	13 - 16
4: Control of infection - sources and dispersal	16 - 17
5: Control of infection - environmental transmission	18 - 21
6: Control of infection - colonisation and infectivity	21 - 22
7: Hospital pathogens.	22
8: Epidemiological methods.	22 - 25
9: Significance of airborne infection.	25 - 27

### 3. Report.

3.1. Aerosol experiments.	23 - 42
3.2. Ultraviolet irradiation.	42 - 46
3.3. Applied Air Hygiene - ventilation of operating theatres ; disposal facilities in wards ; staphylococcal contamination of ward air.	46 - 57
3.4. General discussion.	57 - 58
3.5. Conclusions.	59

### 4. Appendix.

Tables 1 - 39,  
Figures 1 - 17,  
References, and  
Acknowledgements.

# 1 : INTRODUCTION

## 1.1. Title.

The thesis is entitled "Air Hygiene in Hospitals".

## 1.2. Purpose.

In preparing this thesis, I set myself three objectives:

1. to review current concepts of airborne infection in hospital,
2. to record investigations I have made of certain aspects of such infection, and -
3. to consider future developments in the sphere of air hygiene in hospital.

## 1.3. Format.

Each of my several studies represents a separate entity for consideration, although all of them are related to a single topic - air hygiene in hospitals. They are recorded and discussed separately but I present a short discussion to connect the parts with the whole. Tables, graphs and diagrams are gathered together in the Appendix.

## 2 : REVIEW

### 2.1. Historical Review.

#### 1. Before 1900.

Florence Nightingale and Joseph Lister are popularly given credit for evolving the concepts of infection acquired in hospital. The importance of their observations cannot be decried especially when these are assessed in the context of the conditions prevailing in hospitals during their era - the mid-19th Century. However, the elements of the situation were appreciated almost a century before (Solwyn 1966) and the modes of transfer of infections generally were recognised even earlier. (Wells 1955; Riley & O'Grady 1961)

It is salutary to realise that planned ventilation for hospitals as a means to control spread of infection within these institutions was first advocated in 1752. By the time of Nightingale and Lister, not only was planned natural ventilation a reality but examples existed of mechanical<sup>1</sup> extract ventilation and of an elementary form of air-conditioning. (Nuffield Provincial Hospitals Trust 1955) Indeed, during the same Crimean War in which Miss Nightingale earned her fame there was built a remarkable military hospital which included among its many novel features a system for positive-pressure ventilation of wards. (Weeks 1963) It is significant that such ventilation was provided to exclude smells and apparently not to deal with airborne microbes.



Some years before that war, Sir James Simpson, a noted surgeon, stated that "the vitiated air of an hospital" was "full of living spores and germs" which, "when they find a proper nidus, lead on by their development to fermentations, putrefactions, suppurations.....". (Selwyn 1966) Not until after the war was the truth of Simpson's statement proven when, in 1861 Louis Pasteur demonstrated the existence in air of such germs. Pasteur's discoveries prompted Lister, then practising surgery in Glasgow, to conceive the methods of antiseptic surgery with which he associated the use of a carbolic acid spray to disinfect the air of the operating theatre. (Guthrie 1965) A marked reduction in the incidence of post-operative infections accrued from the introduction of Lister's methods but subsequently this was overshadowed by the more dramatic results obtained with the introduction of steam sterilisation for surgical instruments and the application of aseptic surgical techniques.

The apparent eclipse of Lister's methods caused many persons to attribute greater importance to spread of infection either by contact or by ingestion, than to spread through air. This interpretation seemed to be confirmed when Flügge demonstrated that exhaled droplets containing respiratory parasites were of such a size that gravity caused them to sediment out rapidly from aerial suspension. (Wells 1955)

## 2: Between 1900 and 1950.

During the first quarter of the 20th Century, the concept of aerial transfer of micro-organisms virtually fell into disrepute. Flügge droplets, as they were termed, came to be regarded merely as special vehicles for the spread of infection

by direct contact. Despite established scepticism, however, interest was rekindled when Meloney and Stevens in the United States of America reported the instance apparently of aerial transfer of pathogenic streptococci in an operating theatre from the nose of an unmasked surgical attendant to the incisional wound of a patient. (Howard, J.M. et al. 1964)

Initially, investigations centred on the streptococcus, on the operating theatre and on dispersal from the respiratory tract. Gradually, a less restricted attitude developed especially with the work of William Firth Wells and his colleagues, and with that of his fellow American, Deryl Hart. Over a period of some 25 years, Wells developed the hypothesis of "sanitary ventilation" - that is, the desirability of cleansing the atmospheres in which human beings might be exposed to harmful airborne microbes. Wells' principle contribution to the fledgling science of air hygiene was to demonstrate the existence of droplet nuclei. These were the residues of respiratory droplets which had undergone evaporation. Although they were smaller than Flügge droplets, they often contained parasitic microbes and were widely scattered through the atmosphere before being inhaled by a new human host. Another result of these important studies was the production of the first device for volumetric sampling of air. The "traditional" method of air sampling had been developed by Petri before 1900. This procedure was to expose solidified nutrient agar, contained in a Petri dish, so that airborne bacteria-carrying particles could sediment onto the culture medium and, after a period of suitable incubation, would yield visible colonies to be counted and identified. The technique had much to recommend it and continues in use but its crucial limitation is that it collects only the larger

airborne particles which can sediment out under normal conditions of routine use. In essence Wells' device accelerated the effects of natural gravity. A volume of air was drawn into the sampler where the particles in aerial suspension were subjected to centrifugal force - virtually, an artificial and enhanced form of gravity - in order to deposit them on a sampling medium. For the first time, therefore, the concentrations of bacteria-carrying particles actually in aerial suspension could be estimated. (Wells 1955)

Next, a contemporary of Wells, explored the practical implications of disinfecting air by irradiation with ultraviolet light. During the course of these investigations, he observed that the air of many operating rooms was heavily contaminated with staphylococci and that increases in the concentration of these airborne micro-organisms, particularly during the winter months, seemed to be paralleled by increases in the incidence of severe staphylococcal sepsis. Furthermore, he noted that the general content of airborne microbes in a room varied approximately in proportion to the number of persons in the room, the duration of their stay, the degree of their activity or their talking, and the presence of contaminating organisms in the nose or throat of each person. The result was that, even when a supply of clean air was provided to the room, bacteria emanating from the room's occupants rapidly contaminated the air space particularly in the critical zone immediately over the open operative wound. Hart demonstrated clearly that ultraviolet irradiation of the air markedly reduced the concentration of aerial contaminants. But less acceptable was his claim that such aerial disinfection was accompanied by a significant reduction in the incidence of post-operative wound sepsis. (Howard, J.M. et al. 1964)

In Great Britain, the work of both Wells and Hart stimulated interest in the study of air hygiene. This led to the development of a second device for the volumetric sampling of air -- the slit-sampler. However, the advent of war diverted the attention of investigators in both Britain and America away from air hygiene in hospitals and towards the problems of airborne infection in overcrowded military and civilian quarters. The realisation that microbiological warfare was a possibility gave further impetus to research which ultimately yielded much valuable information as well as several new devices and techniques. The results of many wartime investigations did bear upon problems encountered in air hygiene for hospitals. (Bourdillon, Lidwell and Lovelock 1948) But, in the hospital sphere in Britain at this time the greatest impact was made by the decisive demonstration of the benefits obtainable with artificial ventilation of burns' dressing-rooms. (Bourdillon and Colebrook 1949)

### 3: Between 1950 and 1966.

Hart's early appreciation of the emergence of the staphylococcus as the predominant causal agent of infection acquired in hospital was fully confirmed. What he could not foresee was the adaptability of the organism in its efforts to survive in the hospital environment after the widespread introduction of antibiotics for clinical use.  $\beta$ -Haemolytic streptococci, previously the major intramural pathogens, were almost invariably sensitive to penicillin and thus were easily eliminated from the hospital's environment. In contrast, many strains of Staphylococcus aureus soon developed resistance both to penicillin and to many of the antibiotics introduced to supplement it. Apparently these staphylococcal strains

also proliferated to fill the ecological vacuum resulting from the removal of streptococci. So-called "epidemic" strains were identified and were thought to possess unusual powers of infectivity. In fact, this epidemic propensity probably reflected both the organisms' inherent ability to survive in the environment and its acquired ability to resist the effects of antibiotics. Concurrently, there was an increase not only in the numbers of persons receiving treatment in hospital but also in the numbers of patients whose illness or treatment rendered them particularly susceptible to pathogenic microbes.

Methods for identifying different types of staphylococci were developed and were significant in facilitating study of the mechanisms of hospital infection. Much attention was paid, on both a practical and an experimental basis, to unravelling the intricacies of particular facets of the problem. In the field of air hygiene this involved identifying sources of pathogens, elucidating the mechanics of dispersal, demonstrating the vehicles of transmission, and characterizing the attributes for survival and infectivity. Most investigations were of a purely qualitative nature, a few involved quantitative estimations but only rarely was there a serious attempt to assess the inter-relationship of several individual components under study. Frequently, also, the results of studies conducted during epidemic outbreaks were applied by inference to defining measures of control not truly justifiable for more normal non-epidemic situations. Conflicting theories were propounded, new techniques were developed and several old methods were refined. Latterly, the development of typing procedures for various Gram-negative bacilli heightened the growing



awareness of the significance of hospital infections caused by organisms other than staphylococci. Additionally, the first tentative steps were taken to define criteria and establish generally applicable methods for routine surveillance of all types of infections acquired in any part of the hospital.

## 2.2. Current Principles and Practices.

### 1: Airborne Infection - Sources and Dispersal.

The primary source of most micro-organisms pathogenic for one human being is usually another, or the same, human being. - (Fig. 1.) This generalisation does oversimplify the complex relationships which exist in reality. Yet, nowhere more than applied to infection acquired in hospital does it serve to emphasise the most important concept - that the existence of microbial pathogens in an institution is the direct consequence of the presence of contaminated human beings in that building. (Robertson 1958; Barber & Warren 1962)

Both healthy and diseased humans may act as sources for a variety of pathogens. Use of the term "source" infers that the human possesses a site in or on his body where the bacteria may live and multiply. Probably the healthy carrier, devoid of signs or symptoms, is more important than the clinically obvious, diseased patient. However, to be effective as a source for aerial transmission of its organisms, a site must offer opportunities not only for multiplication but also for liberation. It is simple to deduce that sites on the surface of the body will favour

dispersal whereas those situated deeply will require some additional processes if liberation of their microbes is to be accomplished successfully.

Two parts of the body are naturally endowed with mechanisms for dispersal and thus can be considered immediately as regular sources of airborne bacteria. The respiratory tract's propensity in this respect has been realised for years. More recently, the potential for dispersal has been appreciated as an incidental to another well-known physiological process - the continual shedding of skin scales from the surface of the body. In health, Both these sources normally carry commensal bacteria but significant numbers of persons in the community may carry potentially pathogenic bacteria in the naso-pharynx (Lemon, Loosli & Hamburger 1948) or on the skin. (Hare & Ridley 1958) A contaminant commonly present on both these sites is Staph. aureus.

The normal bodily functions of respiration (Baid, Lidwell & Williams 1956) and skin-scale shedding (Davies & Noble 1962) are accompanied inevitably by dispersal of these bacterial contaminants, the numbers of particles dispersed being related approximately to the degree of activity at any particular time. (Austin 1965) For example, coughing and sneezing produce more bacteria than do quiet respiration and normal talking. (Buguid 1945) Similarly, washing, (Hare 1963) showering, (Speers, Bernard, O'Grady & Shooter 1965) and exercise (Bethune, Blevens, Parker & Pask 1965) yield more aerial contaminants than do sedentary movements. However, the extent of bacterial dispersal seems to be related less to variations in the size or number of particles released but more to the concentration of microbes available at the source for contamination of each particle. (Buguid 1946; White 1961;

Ehrenkranz 1964, Noble & Davies 1965) In the diseased state, too, this quantitative difference is detectable in that gross contamination of the source is usually associated with aerial concentrations well above average for the particular contaminants being dispersed. (Rountree & Beard 1962)

This relationship has been observed particularly with certain types of patients - those with "open" respiratory tuberculosis, (Riley et al. 1962) with staphylococcal pneumonia, (Shooter, Griffiths, Cook & Williams 1957) with respiratory virus infections epincidental to nasal carriage of Staph. aureus, (Eichenwald, Kotsevalov & Fasse 1960) with staphylococcal pyoderma, (Solberg 1965) and with a variety of skin diseases. (Noble & Davies 1965) However, these instances of gross dispersal reveal more than just accentuations of normal processes of liberation.

Dispersal into air from other sites is achieved indirectly since the contaminating bacteria pass to an intermediate site from which the actual liberation is effected. Most civilised human beings spend the greater part of their lives in close contact with fabrics, in the form either of clothes or of bed linen. It is not surprising to find, therefore, that these constitute the commonest intermediaries for dispersal. (Hare 1963) Of course, the presence of fabrics which are both clean and closely woven will interfere with the process of natural dispersal from skin. (Bernard, Speers, O'Grady & Shooter 1965; Blowers & McCluskey 1965) This is the rationale for providing special garb for surgical staff but if the clothes or bedding are exposed for only some 48 hours to the natural output of skin, respiratory tract and other bacteria, they rapidly accumulate a high concentration of contaminants. (Walter & Kundsia 1960; Blowers & Wallace 1955) Gross

contamination of fabrics, such as infants' napkins, wound dressings and the bed linen of incontinent patients, is both commonplace and obvious, yet, similar though less recognisable degrees of contamination are derived from patients with extensive skin lesions, (Selwyn 1965) with burns (Bourdillon & Colobrook 1949) and with empyema. (Laurell & Toth-Gyulai 1961) Dispersal from these sites depends upon mechanical disturbance of the fabric (Rubbo & Saunders 1968) particularly during the movements of the clothed body, (Duguid & Wallace 1948) the agitation of bed linen when a bed is made up, (Michaelson 1964; Wysham et al. 1957) and the handling of fouled dressings or bedding. (Thom & White 1962; Church & Leosli 1958) Fabrics have also been demonstrated experimentally as disseminators of vaccinia virus. (Sidwell, Dixon & McNeil 1960)

## 2. Airborne Infection - Environmental Transmission.

Separation of the bacteria from their human, donor host either by direct dispersal or by transfer to another site immediately deprives them of their optimal conditions for survival, let alone for multiplication. They are introduced into the essentially rigorous conditions of the hospital environment where they are exposed to many injurious influences. Rapid evaporation affects them both in air and on surface, (Ferry, Brown & Damon 1958; Hinton, Maltman & Orr 1960; McBade, Hall & Street 1964b) Experiments with virus aerosols suggest that certain viruses, including vaccinia and influenza, are less susceptible to drying out. In contrast, poliovirus survives better in a humid atmosphere. (Harper 1961) But, if desiccation is prolonged, not only may pathogenicity be lost but the vital ability to colonise may be impaired. (Rountree 1963; Hammelkamp et al. 1958; Arsoni & Vasiloglou 1964)

Exposure to light (Lidwell & Lowbury 1959) and to ambient temperature (McDade, Hall & Street 1964a) are also deleterious so that only those organisms able to adapt quickly in response to an ever-changing variety of environmental conditions will survive. Bacteria which can form spores have an inherent advantage in this respect and can be found in many sites in hospital (Gye, Rountree & Loewenthal 1961) although that most commonly found, Clostridium welchii, probably originates either outside the hospital environs or from the gastro-intestinal tract of humans. (Lowbury & Lilly 1959) Of the non-sporing types frequently encountered, Staph. aureus has only a relatively short survival time in dust - less than a week, (McDade, Hall & Street 1963) which is shorter even than the estimated physical half-life of the dust itself. (Lowbury 1959) It is clear, therefore, that the shorter the duration of transfer, the more likelihood there is both of the organism's survival and of its success in colonising a human, recipient host.

The distance over which an airborne organism may be transferred depends largely upon the characteristics of the particle to which it becomes attached at the site for dispersal. The one or two microbial parasites likely to be contained in droplet nuclei, 1-5 microns in diameter, will remain airborne for prolonged periods of time (Duguid 1946) since the effect of gravity on such a small particle is readily overcome by the buoyant influences of air currents. On these airstreams, the particle can travel far from the place where it arose but in such circumstances it can more readily be wafted into the extramural air than be inhaled by a human host and deposited in his lower respiratory tract. (Wells 1955) Larger particles, 6-20 microns in diameter,



remain in aerial suspension long enough only to be projected within the room of origin and therein they settle on any exposed surface. Spread throughout an 8-bedded ward during a period of 48-96 hours has been demonstrated. (Anderson, Coulter & Locke 1960, Rubbo, Stratford & Dixon 1962) This is readily appreciated when it is realised that such particles are easily resuspended for aerial movement by even moderate airflows impinging on the surfaces where they lie. The largest particles, 20-50 microns in diameter, settle quickly only some 5-7 feet away from their point of origin, therefore they constitute the bulk of the dust in hospital. This dust is a mixture of mineral, animal and textile debris, the last being mainly cellulose fibres. (Rubbo, Pressley, Stratford & Dixon 1960) Dust tends to remain on the floor unless forcibly resuspended in air by the violence of some human activity - walking, floor-cleaning, bed-making - but airborne transfer is minimal, so marked is the effect of sedimentation. The average size of particulate matter in the air of hospitals is estimated to be 12-15 microns (Noble, Lidwell & Kingston 1963) and particles such as this have been found to carry on average four viable cocci per staphylococcus-bearing particle. (Lidwell, Noble & Dolphin 1959) Seldom more than 10 cocci have been found although the particles examined were obtained in wards when active spread of staphylococcal infection was absent.

### 3. Airborne Infection - Colonisation and Infectivity.

The final step in this process of aerial transfer involves the organism and a new human host establishing together a host-parasite relationship. This stage in itself is not peculiar to the aerial route of transfer of

infection. Organisms transferred by contact face similar obstacles unless they are ingested. However, it has been estimated that relatively few airborne microbes actually survive to reach a new host. Therefore, colonisation, which is the minimal requirement for growth of the organism, may be difficult to initiate and to maintain in many instances. If successfully achieved, colonisation can develop into a carrier relationship or even into frank infection but to accomplish this the organism has either to breach or to circumvent the natural defenses of its new host. The majority of these interactions between a host's defenses and an organism's infective powers are also common to all other host-parasite relationships. Therefore, I will restrict further consideration to the mechanisms for colonisation from air.

In health, the skin is intact thus effectively excluding environmental bacteria although these may land on it and colonise it, especially in the "wetted" areas where abundant secretions favour establishment of a carrier state. Localised infections of hair follicles and of minor cuts and abrasions are frequent sequelae of such surface colonisation. Experimentally induced infections have been produced with as few as 15 organisms per inoculum although in this instance multiplication was most rapid when the lesion was covered. (Poster & Hutt 1960) However, any artificial opening into, or loss of, skin provides a unique opportunity for the ingress of bacteria. These may arise locally from adjacent skin or may be implanted from air, instruments, sutures or other contaminated vehicles of transmission. Few surgical incisions have been found to be entirely free from contamination prior to closure of the skin. A considerable proportion of them

contained Staph. aureus strains identical to those isolated from sites elsewhere on the patient's body. But an almost equal number yielded several different strains corresponding to types isolated concurrently from the air. Less often there was a correlation between types obtained from the wound and those isolated from the surgical staff. (Burke 1963; Ives & Hirshfield 1938; Wise, Sweeney, Haupt & Waddell 1959) In view of these findings, it is almost surprising that so few surgical wounds do develop sepsis due to such bacteria.

The area exposed during an operation and the duration of exposure has been postulated as being of significance in influencing the degree and type of contamination. One estimate indicates that an average wound exposes an area of 12 square inches directly to the sedimentation of airborne bacteria. Indirectly, however, through transfer of sedimented bacteria from surgical gowns, drapes, instruments, gloves and wash-bowls, a catchment area of some 432 square inches is available throughout the duration of the operation for the sedimentation of bacteria which could threaten the wound. (Devenish & Miles 1939) It has been suggested that the placing on the list of operations may also be significant in ensuring either a clean or a contaminated environment, (Stewart & Douglas 1962) but more importance seems likely to attach to quantitative differences between inocula. In this context, it has been shown that quite small numbers of bacteria on suture materials have induced appreciable inflammatory reactions whereas wound-closure with "butterfly", sticky-tape strips carried out at the same time did not cause any inflammation. (James & MacLeod 1961; Caspendale & Sereda 1965)

The other common site of deposition for airborne bacteria is the respiratory tract. Droplet nuclei pass unimpeded through the upper part of the tract and are trapped in the small air passages in the lower part. It is now accepted that even an inoculum of one organism deposited in this way may initiate infection, the tubercle bacillus being the example which springs most readily to mind.

(Hiley et al. 1962) However, less is known about the fate of bacteria conveyed on the larger particles which are trapped by the ciliated epithelium of the upper respiratory tract, especially in the naso-pharynx. The phenomenon of bacterial interference has been demonstrated (Boris et al. 1964) whereby implantation of an apparently non-pathogenic strain of Staph. aureus on the nasal mucosa has prevented subsequent colonisation with pathogenic strains. In another experiment, rejection of experimentally inoculated strains of Staph. aureus on the basis of an immunological response, was postulated to explain inability to colonise the nasal mucosa of certain persons. Also observed was the fact that a certain minimum inoculum was required merely to initiate colonisation on account of the non-specific inhibitory effect of the resident flora - possibly another facet of bacterial interference? (Phrenkraus 1966)

#### 4: Control of Infection - Sources and Dispersal.

In themselves, the efforts to identify and to define the elements of hospital-acquired infection would be mainly of academic interest if it were not for the existence of an important underlying purpose - the need to protect the health of the hospital's patients by determining how, when and where to apply measures to interrupt the "life-cycle" of the infection.

Research and prevention are frequently complementary in practice because knowledge of this "life-cycle" is fragmentary as yet, particularly with respect to the environmental sojourn of the micro-organisms involved.

Measures to reduce dispersal from human sources have been applied for many years especially in the operating theatre although refinements in methods are introduced at intervals. Thus, the provision of suitable surgical masks (Shooter, Smith & Hunter 1959; Thomas 1961; Greene & Vesley 1962) and of properly designed theatre clothing (Bernard, Speers, O'Grady & Shooter 1965; Blowers & McCluskey 1965) is essential to ensure that naturally released bacteria are contained during surgical procedures. Other measures to deal with contaminants at their human source include the isolation of patients known to carry or to be infected by organisms with epidemic propensities (Williams et al. 1962) or the segregation of post-operative patients from those awaiting operation. (Shooter et al. 1963) Suitable facilities and adequate discipline are essential if these methods are to succeed. In their absence, other methods have been adopted which are based mainly on the use of antibiotics to eliminate the organisms at their site of colonisation - the nose, (Elek & Fleming 1960; Cope, Shooter, Green & Noble 1961; Barber & Warren 1962; Rountree & Beard 1962) the umbilicus (Gillespie, Simpson & Tozer 1958) or the skin. (Selwyn 1965) The obvious risk with this method is that frequent and prolonged exhibition of a particular antibiotic may result in the development of resistance to it by the predominant pathogens in the hospital, principally the Staph. aureus. (Jevons, Coe & Parker 1963)



### 5: Control of Infection - Environmental Transmission.

The second opportunity for action presents itself when the organisms are present in the environment. Elimination of environmental reservoirs can best be achieved in new buildings by careful planning. (Sovic, Tonkin, Robson & Dixon 1964; Bagshawe 1964) However, a plan is only as effective as the knowledge upon which it is based thus many new hospitals either perpetuate old faults or incorporate new ones. In existing departments, the presence of microbial reservoirs may be countered by disinfection - of bedding (Marsh & Rodway 1954; Bowers & Wallace 1955; Coplan 1962), of the ward structure and its associated fabrics (Ayliffe & Beard 1962) or of apparatus in which aerosols may be generated. This last group embraces a wide variety of equipment including the humidifier units of both air-coolers (Anderson 1959) and babies' incubators, (Hoffman & Finberg 1955; Sever 1959; Becker 1962) and the atomizers of inhalation-therapy devices. (Macpherson 1958; Reinerz, Pierce, Mays & Sanford 1965) Additional or alternative measures may be taken to contain the dispersal of microbes, among them being bagging of used linen, (Huret, Grossman, Ingram & Love 1958; Rogers & Slater 1961; Dokinsky 1962; Michaelson 1965) mechanical sluicing of fouled linen, (Sandiford, Bowers & Mitchell 1959), oiling of floors and bed linen, (Lemon, Locali, Wise & Puck 1952) vacuum-cleaning of carpets (Shaffer 1966) filtration of air discharged from surgical suction apparatus and from vacuum cleaners, (Bowers, Mason, Wallace & Walton 1955; Bate 1961; Bowers & Bound 1960) ventilation or enclosure of water-closets (Darlow & Bale 1959; Bound & Atkinson 1960) and the use of oiled mops for floor-cleaning. (Kingston 1963; Dabb, Lilly & Lowbury 1963) Indeed a variety

of procedures to combat an equally varied selection of dispersal processes although probably the most important procedure, and that most often neglected, is to discipline the activities of the hospital's staff. (Kethley 1964)

Environmental control can also be effected when the microbes are actually airborne. The obvious choice in this instance is ventilation although the lack of control over natural airflows does not favour the use of natural ventilation in areas of importance. Thus artificial ventilation has been widely advocated for operating theatres (Sevitt 1958; Blowers, Mason, Wallace & Walton 1955; Shooter, Taylor, Ellis & Ross 1956; Wolf, Harris & Dyer 1959; Lidwell & Blowers 1962) but less often for general wards. (Steingold, Dunn, Hawksworth & Limb 1963; Ayliffe & Beard 1962) This advocacy has been qualified especially when a reduction in the incidence of post-operative infection has been sought but not obtained in association with a reduction in the operating theatre. (Kinmonth et al. 1958; Howe & Marston 1963) Concurrently, there has been considerable controversy over the relative merits of two types of ventilation - downward-displacement and turbulent. The former removes, with minimal dispersal, bacteria originating from sources below the level of the operating table whereas the latter is more effective for sources above the table because it carries bacteria away from the wound by turbulent mixing of air. Each has advantages although the desired downward, displacing airflows of the first system are readily counteracted by convection currents flowing upwards from such sources of heat as the human body and the theatre lights. More recently, however, the development of ultra-clean rooms for industrial use has been accompanied by the introduction of a third ventilating process depending upon a streamlined or laminar airflow pattern at air-change rates far in excess

of the 10-25 changes per hour used for the conventional "dilution" systems. Such laminar airflows sweep away all particles larger than 0.3 micron diameter so rapidly and with such control of direction that the alleged deficiencies of both of the conventional processes are obviated. However, for benefits to be obtained from this, the operating room must be re-organised so that the flow of clean air passes first over the area for maximal cleanliness - the instrument tables, positioned at the patient's feet. Thereafter, it becomes progressively more contaminated as it flows past the wound and the surgical team, the patient's head, the anaesthetist and any unscrubbed staff, before being removed from the area of minimal cleanliness - the changing and washing rooms for the staff. (Eccleston 1964; Beck 1966)

The principle of high air-change rates has also been applied to a pressurised, plastic operating enclosure, to provide a particularly clean environment within an ordinary operating theatre. (Charnley 1965; Levenson, Trexler, La Conte & Pulaski 1964)

However, the principles and practices which are generally acceptable at present, relate to particle sizes of 5 microns or larger and to air-change rates not exceeding 25 per hour.

Aerial disinfection by ultraviolet irradiation has achieved some popularity for use in operating theatres particularly in America, (Howard J.M. et al. 1964; Bernard, Speers, O'Grady & Shooter 1965) and to a lesser extent for use in wards. (Laurell & Ronge 1955; Stratford 1963b)

Irradiation is commonly employed for disinfecting the special inoculation cabinets in medical laboratories and has also been advocated for use in a cabinet for the dressing of hand wounds. (Sussman, Barnes & Lenihan 1961)

Yet another use has been to sterilise water (Stratford 1963a, Fredette 1963) but on the whole ultraviolet irradiation has not achieved much favour in Britain as a bactericidal agent. Aerial disinfection by bactericidal vapours has received attention but this too has not been used extensively in recent practice. (Kingston, Lidwell & Williams 1962)

#### 6: Control of Infection - Colonisation and Infectivity.

Control of infection may be attempted, finally, at the stage of contact between parasite and new host. The characteristics of a susceptible host cannot be defined fully at present, far less can they be said to be invariable. Extremes of age, debility, exposure to specialised surgery and prolonged or intensive treatment by radiotherapy or chemotherapy are all recognised as predisposing to infection. Small ward units with ventilation planned to exclude aerial pathogens have been built for groups of patients undergoing X-ray therapy (Bagshawe 1964) and renal transplantation (Bowie, Tonkin, Robson & Dixon 1964) although an essential feature of such units has been control of activities as much as control of ventilation, the two being considered to be complementary. Chambers for the isolation of individual patients with extensive burns have been developed with positive-pressure ventilation to exclude airborne pathogens arising from other patients or from staff. Every precaution is taken to minimise contact transfer from outside the isolator but it is significant that there is a considerable risk that the patient may infect his lesions from his own sites of bacterial carriage, especially if these are within hands' reach. In contrast, a simple system with a similar purpose has been described whereby only the burned portion of the body is actually

isolated and exposed in sterile air. (Potter 1964)

### 7: Hospital Pathogens.

In the foregoing paragraphs, I have attempted to outline a representative selection of the many facets of air hygiene in hospital. However, the review would not be complete without some remarks on the types of organisms commonly involved and the methods available to isolate and to identify them. Although the Staph. aureus has assumed the role of pre-eminent intramural pathogen in hospital, the undue emphasis which it has received has obscured the fact that other pathogens, principally Gram-negative bacilli, exist in the hospital environment. These bacilli are less predictable than the staphylococci regarding sensitivity to antibiotics and frequently they do not develop, as a population, any uniform pattern of resistance. Additionally, they rapidly manifest marked changes in resistance. The staphylococci survive in dry conditions and therefore appear relatively frequently in air, as too do Cl. welchii, but the Gram-negative bacilli are orientated more towards moist conditions and are present in air only infrequently. In this respect, it is significant that such bacilli, particularly of the *Pseudomonas* and *Proteus* species, were reported as the main contaminants both in the humidifiers of air-cooling units and of infants' incubators, and also in the atomizers of inhalation-therapy equipment.

### 8: Epidemiological Methods.

Undoubtedly, the epidemiological investigation of staphylococcal infections in hospital has been facilitated by the availability of bacteriophage typing as a routine

procedure for identifying particular strains of Staph. aureus. (Blair & Williams, 1961) Until recently, any similar differentiation of strains of Gram-negative bacilli was not possible thus little value was to be derived from attempts to unravel the complexities of cross-infection attributable to such organisms. The desired techniques are now available and already several investigators have shown interest in the problems involved. Colicine typing has served to identify strains of Escherichia coli related to cross-infection in a urological ward (Linton 1960) although serological typing of the "enteropathogenic" strains of this organism has been established for some time. (Rogers & Taylor 1961) Phage-typing of Salmonella organisms has been available for some time also (Anderson & Williams 1965) but the methods have now been adapted to yield a routine typing system for Pseudomonas strains. (Sutter, Hurst & Fennell 1965) Additionally, Pseudomonas organisms can be typed by a combined serological and pyocine method (Wahba 1965) so that only the Proteus group remains relatively undifferentiated except for a rudimentary division possibly on the basis of Bienes' phenomenon. (Story 1964) These techniques are not restricted to the study of air hygiene, of course, although this does benefit from their availability as much as any other epidemiological study of a similar nature. In contrast, the sampling devices are very characteristic of air hygiene work and an excellent summary of the features and applications of most types has been published. (Wolf et al. 1959) I have described two types briefly already, in the historical review, although only the settle-plate continues to enjoy widespread acceptance and use, despite its limitations. The other air-sampling technique with which I am familiar is use

of the Bourdillon, or Casella, slit-sampler. Therefore, I will confine my remarks to describing this device.

The slit-sampler consists of an air-tight chamber which encloses a motor-driven turntable large enough to accommodate a 3 $\frac{1}{2}$ " or a 6" (90mm. or 150mm.) diameter Petri dish containing a suitable bacteriological sampling medium. Located in the top of the chamber above the turntable is an air-inlet tube of which the lower end is closed, except for a slit, 0.83mm. wide which is positioned radially to the centre of the turntable. The turntable, with a Petri dish in position, can be raised or lowered mechanically so that the underside of the slit and the upper surface of the agar sampling medium are at a pre-determined distance apart. Thus, when a vacuum is induced in the chamber, air flows rapidly through the slit from the atmosphere so that any bacteria-carrying particles are impacted on the agar which is revolving slowly beneath the slit. After a period of suitable incubation, the plates are examined and the colonies which have developed can be enumerated and identified as required. The apparatus has a uniform airflow rate of either one or five cubic feet (28.3 or 141.5 litres) per minute, the total volume of air sampled being determined by the duration of sampling. This latter, in turn, is pre-set by selection of appropriate combinations of gears used to transmit the motor-drive to the turntable - one revolution of the table occupying half a minute, two minutes or five minutes. The device permits relationships to be established on a basis of time, between varying concentrations of airborne contaminants and particular environmental events. For this, it is invaluable especially as a tool for research, but it does not differentiate the sizes of the particles it collects.



A modified design for this purpose has been described (Lidwell 1959) but the only commercially available size-grading sampler is the Anderson cascaded-sieve which is not so suitable for time-concentration studies. (Anderson, 1958)

Sampling of both surfaces and textiles has been advocated as a complementary procedure to air sampling in any serious attempt at a comprehensive air hygiene investigation. The techniques are in process of being refined for routine use and although I have not used them, I will make passing reference to them. Surfaces were sampled on a purely qualitative basis prior to the development of a reliable, semi-quantitative technique. (Walter & Kundsia, 1960) Truly quantitative methods are now available in the form of the bandage-reinforced agar-slab impression process (Foster 1960) and the "Redac" plate technique. (Bond et al. 1963) Similarly for sampling textiles, a semi-quantitative assessment of the numbers of contaminants available for dispersal was obtained with Williams' "sweep-plate" (Bowers & Wallace 1955) but the "percussion-plate" (McGuade & Sutherland 1960) and the "friction-dispersal" technique (Rubbo & Saunders 1963) established standardised conditions for assessment.

### 9: Significance of Airborne Infection.

With the accumulation of knowledge and the refinement of techniques, there has come a growing realisation that isolated studies of essentially epidemic situations, conducted according to differing criteria and with a variety of equipment, media and methods, cannot yield generally significant results. This is particularly true of attempts to assess the meaning

of variations in bacterial concentrations in air at different sites and at different times. Without a "normal" value as yard-stick, the relative importance of each observation cannot be assessed realistically, but only empirically. Yet, often the bacteriologist is asked to recommend control measures on the basis of such an assessment only to find his proposals condemned as unduly complicated or expensive - cannot something simpler or cheaper achieve the same results? The realization of this dilemma has spurred some investigators, particularly in America, to try to establish the required "normal" value by undertaking routine sampling at numerous sites for a prolonged period of time. (Greene, Vesley, Bond & Michaelsen 1962a, 1962b; Shaffer & McDade 1964; Shaffer, Migitt & Key 1965) From their reports, so far, it is clear that bacterial concentrations differ greatly for different hospitals, for different departments in each hospital, for different areas in each department and for different times at each area even with only short intervals between sampling. These observations apply as much to counts of total bacteria as to those of Staph. aureus, there being little relationship detected between the two as the latter often consisted of "showers" of organisms appearing in the absence of a definite source, either human or environmental. Total counts do seem to reflect the standards of housekeeping and of personnel discipline existing in each hospital at any particular time but the unpredictable and gross fluctuations in counts have prompted some observers to suggest that routine air sampling of this pattern is both unrealistic and almost useless. (Warner & Glasco, 1963) Certainly, there is only the most ill-defined correlation detectable between aerial counts of Staph. aureus and incidence of staphylococcal infection

during non-epidemic periods.

In concluding this review of current concepts, I think it worth noting that several publications contain useful contributions on the topic of air hygiene in hospitals, and particularly on its relationship to the problem of cross-infection as a whole. (Dourdillon, Lidwell & Lovelock 1948; Wells 1955; National Research Council of Canada 1960 and 1961; Williams, Bowers, Garrod & Shooter 1960; Riley & O'Grady 1961; Colbeck 1962; Williams & Shooter 1963; Howard, R.S. et al. 1964)

### 3 : REPORT

#### 3.1. Aerosol Experiments.

##### 1: Purpose.

These experiments were undertaken for two main reasons: to study the patterns of decay in concentration and of transfer of airborne organisms within a building subjected to natural ventilation, and to examine certain practical problems which apply to the routine use of slit-samplers.

##### 2: Materials and Methods.

The artificial aerosols used in these studies were produced with a spinning-top, homogeneous spray apparatus (May 1949), made specially to the specifications of the Microbiological Research Establishment. This atomizer consists basically of a small cone which is seated in a housing shaped to accommodate it. Shallow cuts made around the sloping face of the cone form small vanes on which impinge jets of compressed air delivered through orifices cut in the sloping side of the housing. These jets cause the top to spin at a rate which is directly proportional to the pressure of air supplied. A superstructure is provided to support a needle with which to deliver fluid for atomization onto the flat, upper surface of the spinning cone. The fluid is drawn out by centrifugal force into a thin film which fragments at its periphery to form droplets. The droplets are thrown into

the atmosphere in sizes related to the rate of spinning of the top. The final size after evaporation of the fluid of suspension will depend upon the amount of solid matter in each droplet as it was formed. The droplet sizes chosen as appropriate for the experiments envisaged were in the range 12-15u as this is the range of sizes usually found in many hospitals. (Noble, Lidwell & Kingston 1968) According to May's calculations, (1949) a delivery pressure for air of 5 lbs./sq.ins. produced particles of approximately 40u wet size but these rapidly evaporated to a dry size of about 15u.

The test suspension of bacteria consisted of a broth culture of spores of Bacillus subtilis var. globigii, at a strength of  $1 \times 10^7$  organisms/ml.. A stock culture for this was prepared by growing B. globigii in broth for 48 hours before transferring the culture to a +4°C refrigerator. After the culture had sporulated and the spores had settled out, the supernatant fluid was decanted and the deposit was centrifuged to remove excess broth. The spores were then resuspended in water and this suspension was placed in a bath of boiling water for 10 minutes to destroy any remaining vegetative forms. A viable count was performed to estimate the concentration of the spore suspension before the stock culture was diluted to a final concentration of  $1 \times 10^9$  spores per ml., prior to storage at +4°C. When required for use, a suitable quantity of test suspension was prepared on the basis of 0.2cc. stock culture added to 24cc. of nutrient broth, along with 0.8cc. of 5% "Tween 80" which was required as a wetting agent to facilitate the formation of droplets.

The sampling apparatus comprised one or both of two Casella slit-samplers, which have been described already.

The types available accommodated standard 3½" diameter Petri dishes and had a sampling airflow rate of one cubic foot per minute. The gear-drive on each was adjusted to provide a sampling period of 30 seconds for each revolution of a sample dish. Samples were taken onto "Oxoid" nutrient agar which was incubated for 18 hours at 37°C before colonies were counted.

The layout of the building in which the experiments were conducted is shown in the form of diagrams in the Appendix (Fig. 2) along with indications of the siting of the equipment for each of the experiments.

### 3: Experimental Procedures.

Before carrying out any of the formal experiments, it was necessary to undertake several preliminary tests. After its delivery from the manufacturer, the atomizer underwent several trial runs during which its rotational speed was estimated by the use of a borrowed stroboscopic light unit. This device also allowed checking of the speed for uniformity at different pressures of delivery air. The rate of spinning produced with an air pressure of 5 lbs./sq.ins. was found to be constant within acceptable limits although this was assured only if the atomizer was kept scrupulously clean. A short series of tests was performed to estimate approximately the size of the droplets produced by the atomizer at this pressure. For this purpose, a suitable quantity of the test suspension was atomized in a large fume cupboard. Samples were taken by both slit-sampling and sedimentation plates exposed together for periods of 5 minutes. From the pairs of counts obtained, Petri ratios were calculated. Each ratio ( $P_g$ ) is the result of dividing the number of bacteria-carrying particles settling

per square-foot per minute (A) by the number of such particles in suspension in one cubic foot of air. (V) The ratios (A:V) were averaged and the mean value for  $P_a$  of 14.7 was obtained. Reference to a nomogram printed on page 349 of "Studies in Air Hygiene", (Bourdillon, Lidwell & Lovelock 1948) showed that the approximate diameter of the particles produced by the atomizer was 14 microns.

One further series of preparatory tests was performed, principally to determine the quantities of test suspension required to yield numbers of colonies per sample plate which would be countable. These preliminaries proved more useful than was envisaged originally. It was found that 10cc. of test suspension, atomized over a period of 5 minutes, was sufficient to yield suitable colony counts. However, all three operators involved in the performance of the various experiments gave rise to obvious variations in the accuracy of counting colonies. To assess the significance of these differences, each operator counted the colonies on 28 settle-plates on three occasions. The results of this ancillary investigation are considered later. Additionally, it was observed that the atomizer on its own did not project droplets sufficiently far into the atmosphere for them to mix therein and be transferred in adequate numbers to the sampling site. A propeller fan was obtained and, when in operation during the entire period of sampling, was found to improve the performance of the aerosol system greatly.

The formal experiments consisted of 28 separate events of atomizing and sampling, excluding those which proved to be technically unsatisfactory. Samples were taken for the first 30 seconds of each minute, commencing with the time of



initiating atomization. The experiments were not performed more frequently than twice in any morning or afternoon to avoid the risk of accumulating test organisms in the environment. A control plate was always exposed for 30 seconds in the minute before atomization began but this never yielded B. globigii after incubation. The days chosen for the conduct of the experiments exhibited similar features - they were dry, bright and with only light breezes. Dry bulb and wet bulb temperatures were recorded for each experiment and from these the percentage relative humidities were calculated.

Several different experimental arrangements were studied, which can be summarized as follows -

- a. One sampler (W); 3 rooms; 10 sites.
- b. One sampler (R); 1 room; 4 sites.
- c. Two samplers; 1 room; 1 site, but 2 positions, simultaneous sampling.
- d. Two samplers; 1 room; 1 site, simultaneous sampling. with or without tubing attachments.
- e. Two samplers; 3 rooms; 3 sites, simultaneous sampling at 2 sites.

Arrangements c. and d. required that the two samplers were set back to back, equidistant from the atomizer, and with their air inlets 10" apart but at the same height above floor level. When samples were drawn through tubing, the air inlet of the machine with the tube was defined as the mouth of the piece of 1" bore glass tube, 12" long, which was attached to the sampler's own inlet nozzle.

#### 4: Results.

The counts of airborne bacteria-carrying particles sampled at intervals of time after commencement of atomization are tabulated for each experiment. (Tables 1-9, 13, 14, 24 & 25) Also tabulated are the results of the study of counting accuracy. (Tables 18-23)

In the counting experiment, the counts for each plate were averaged to find the "true" count. (Table 10) The frequencies and measures of deviations from each of these means were calculated. (Tables 20-22) The frequency distributions were used to determine the probability with which each operator might be expected to count the "correct" number of colonies. The average deviations calculated for all operators were plotted on log-log graph paper against different values of actual counts. (Fig. 6) From the graph, whole numbers were chosen as "correction factors", appropriate for particular ranges of counts. With these, limits could be defined within which a true count would lie. (Table 23A)

For the experiments involving only one sampler, the individual counts and their range of count values (Tables 1-7) were converted into logarithms to the base 10. These log values were plotted on graph paper as a log-linear plot of a concentration-time relationship. The frequency with which samples were taken of the airborne concentration served to reveal appreciable variations of concentration over short periods of time. But the inaccuracies which could be attributed to counting rendered it difficult to assess the significance of these variations. To simplify interpretation of the graphs, all plotting was done on large-scale sheets which cannot be reproduced in this thesis. Tracings of small-scale replicas of these are included to illustrate

the visible features of the decay-curve patterns under consideration. (Figs. 3-6)

The original intention was to draw the best-fitting straight line, as judged by eye, for each individual plot. The rate of disappearance per hour was calculated in accordance with the equation described on page 62 of "Studies in Air Hygiene", (Bourdillon, Lidwell & Lovelock 1948)

$$K = \frac{(\log_{10} N - \log_{10} n) 138}{t}$$

where K is the rate of disappearance per hour, t is the time in minutes between the moments when aerial concentration N reduces to concentration n, and 138 is a constant for this equation. The K values were to be used in conjunction with visual inspection of the graph plots to compare the features of natural ventilation in the three different areas of the one building. But, when the graphs were plotted they indicated clearly two important observations. Firstly, a straight line could be fitted quite justifiably to some of the plots but others were unsuitable for this treatment because a straight line slope obviously did not reflect accurately the events which were recorded. Secondly, all the curves exhibited in varying degree an initial period of rapid decay which preceded a period of more gradual decay. Three examples from the large-scale graphs have been reduced in scale to illustrate these observations and the combined tracings are included in the Appendix. (Fig. 7) Further consideration of the decay-curves was based solely on comparisons of their visible characteristics.

Inspection of the four sets of principal graph-plots (Figs. 3-6) reveals certain basic features. As might be expected with atomization of a uniform quantity of bacterial

suspension over a constant period of time, the maximum aerial concentration achieved differs with the volume of the room in which atomization took place. But it does not show much variation for different sites in the same room. This is most noticeable when experiments were performed during the same morning or afternoon as was the case with sampler W in the small ward, runs 1 and 2, (Fig. 3) with the same sampler in the large ward, runs 3 and 4, (Fig. 5) and with sampler R in the small ward, runs 1 and 2. (Fig. 6) The variation relates both to the time of appearance and to the value of the maximum concentration. The most rapid increase and the highest concentration both develop in the duty room (2300 ft<sup>3</sup>) with a less rapid increase and a lower concentration appearing in the small ward (11,160 ft<sup>3</sup>). The lowest values apply to the large ward (22,325 ft<sup>3</sup>).

Inspection also reveals that each curve, as well as showing the feature of initial rapid decay, already mentioned, also tends to assume a biconcave shape with the concavity upwards in its early stage of decay but downwards thereafter. This appearance is most noticeable when the principal pattern of decay is gradual (Fig. 3) but it is hardly discernible when this is rapid. (Fig. 6) Rapid decay seems to be associated also with very few fluctuations in concentrations. In contrast, some of the variations observed with gradual decay amount to small but actual increases in concentration which then serve as fuel for new patterns of decay.

The counts obtained with the use of two samplers operating together at one site were not converted to logarithmic form. Interest in them did not centre upon decay-curve patterns but upon the actual relationships of paired counts. Inaccuracy in counting was known to account for at least part of any

difference between pairs of numbers. Thus, counts were not considered actually to differ unless their difference was greater than twice the value of the "correction factor" appropriate for the pair of counts. The frequencies with which these "actual" differences were observed were used to calculate the probability of obtaining comparable air sample counts with the two samplers either without a tubing attachment (Tables 8-12) or with it. (Tables 13-17) The results indicate that for more than half the number of uses, differences between counts obtained simultaneously with the two samplers may be attributable in part or in whole to inaccuracies in counting. The positions of the machines may also be significant in producing differences. When there is a real difference, then sampler B will invariably yield counts higher than sampler W. The interpretations are valid only for samples drawn directly from the atmosphere. In contrast, when a short length of straight tubing is interposed vertically between the air to be sampled and either of the samplers, it consistently lowers the counts obtainable with the machine to which it is attached.

The results for the series of experiments to assess aerial transfer within a building were not converted from real figures, either, because here too the purpose was to examine actual events and not rates of decay. (Tables 24 & 25) The paired sets of counts should be examined in conjunction with the appropriate diagrams (Fig. 2) which present all the relevant environmental information in summarized form. The first two pairs of results demonstrate clearly that the majority of airborne bacteria will travel with prevailing airflows but not against them. Also, they show that any structural feature which serves to increase

airflow - in this instance, an open doorway - will increase the transfer of bacteria. It must be noted that a few bacteria do seem to flow backwards against the current. By the use of smoke to visualise the actual airflows associated with doorways, it was possible to detect small peripheral flows of turbulent air which passed in the opposite direction to that of the major flow. Often, thermal differences across the doorway induced this transfer of air in one direction at the top of the door and in the other at the bottom. The second two pairs of results show that appreciable numbers of airborne bacteria may pass not just from one room to the next but on again into a third room in a relatively short period of time if the strength and direction of airflows is favourable. But the existence of turbulent counter-flows will almost invariably result in a few bacteria appearing upstream of the principal flow of air. This appearance is not related to gaseous diffusion and must not be confused with it.

### 5: Discussion.

Aerosols have been used on several occasions to demonstrate the efficiency of ventilating processes. (Wells 1955; Bowers & Grew 1960; Kethley 1964) If these processes are controlled, as is the case with artificial ventilation, then it has been shown that an exponential relationship for decay does exist. But in the absence of controls, several different decay-curve patterns may emerge either with one pattern predominating or more often as a mixture of patterns. The uncontrolled behaviour of natural ventilation is well known and probably is the cause of the marked variations from

a straight-line relationship observed during the experiments reported here. It might be reasonable to adduce that this very turbulence would ensure homogeneous mixing of an aerosol in the atmosphere. But, the results obtained with paired samplers operating at one site suggest that quite marked differences in aerial concentrations probably exist at any given moment of time at each of a large number of sites in a room.

The appearance of a period of rapid, initial decay in the life of artificial aerosols has been reported (Darlov, Powell, Bale & Morris 1958; Ferry, Brown & Damon 1958) but in these instances the aerosols were contained within controlled, artificial environments. This "initial kill" was attributed to two principal influences. Partly, it is due to mechanical destruction of bacteria in the process of atomization in consequence of the use of force to accomplish the formation and projection of droplets. But predominantly, it is the result of rapid evaporation which has a lethal effect upon bacteria in the droplets as soon as these become airborne. Many organisms, especially the bacilli, are susceptible to such rapid changes in their micro-environments. The published reports of this phenomenon of "initial kill" related to aerosols which were produced and sampled under the conditions of a laboratory experiment. The observations recorded here demonstrate that similar effects will influence the patterns of decay of atomized contaminants in a natural environment. It has been suggested that a more realistic and less violent method of "atomization" would be preferable for simulating natural dissemination. One procedure which has been described depends upon artificial contamination of a fabric with a known number of organisms in liquid suspension.



Time is allowed for the bacteria to adjust to the environmental conditions before the fabric is agitated and the microbes dispersed into the atmosphere. (Bubbe, Stratford & Dixon 1962) The method avoids both the disadvantages of the more usual yet forceful procedures of atomization. Its very simplicity is attractive and for purely qualitative experiments it has been proven satisfactory. But for quantitative estimations it might be inadequate since the conditions for drying and for dissemination would be difficult to standardise.

Effects due solely to artificial processes which do not have natural counterparts must be excluded when results obtained with tracerbacteria are assessed in relation to a "real" situation. Usually, it will suffice to determine from a graph plot of the values observed the duration and extent of the "initial kill". This portion of the decay may be omitted when the rate of disappearance of aerial contaminants is calculated as a log-linear relationship. But similar values for rates of decay can apply widely to separated groups of concentrations. Thus, a false impression will be imparted if only the rate of decay is quoted and the point of origin of each decay-curve is not defined. In practice, the benefit to be derived from ventilation is not determined solely by the rate with which it removes bacteria from the atmosphere but almost equally by the magnitude of the aerial concentrations which require to be removed. In the causation of infection, the important measure is the product of the average concentration available and the duration of exposure to that concentration. (Lidwell 1963; Kethley 1964; Roach 1966)

The maximum concentration, and the rapidity with which

it is attained, is determined by two counteracting effects. On the one hand, the rate and the duration of generation of airborne particles regulates the numbers of organisms entering the atmosphere. On the other hand, as soon as bacteria-carrying droplets enter the atmosphere they are subjected to both biological and physical decay. The observed accumulation of aerosol represents the combined effects of these processes at any specific moment of time. Initially, the rate of generation exceeds that of decay and there is a detectable increase in aerial concentration. Gradually this tends towards an equilibrium where the two rates balance each other. A constant concentration in air would be the discernible result if atomization thereafter was a continuing process. However, in reality atomization is seldom continuous thus at some stage the decay effects come to predominate and it is these which are enhanced by ventilation or by the use of bactericidal vapours. But knowledge is required of both stages of the dispersal and decay cycle before these enhancing effects can be applied in practice. Therefore, the use of artificial aerosols to simulate the behaviour of natural aerial contaminants must be considered carefully with respect both to the atomizing process and the sampling process.

My observations indicate that at least three of the commonly accepted elements of routine air-sampling may incur a significant degree of error - colony counting; the use of two or more samplers; and the use of tubing during sampling. Little can be done to alter the inaccuracy in counting. Due allowance should be made for its existence when assessing the significance of small differences observed for various situations. Unless the difference between two counts exceeds

twice the value of the relevant deviation to be attributable to counting, there is the possibility that the difference is not a true one. Samplers should not be operated together unless they have been matched during extensive testing in parallel to establish their relative relationships. The existence of detectable differences in arial concentrations located only a few inches apart indicates the need for more than one sampling point in any atmosphere and also confirms the observations of other investigators regarding similar events in reality. (Warner & Glasco 1963; Shaffer & McDade 1964; Kethley & Cown 1966) Even then, only approximately representative estimations of arial contamination in a room will be possible unless some sampling routine based on random selection of sampling sites and conducted over a prolonged period of time can be undertaken. The practical and technical implications of this are self-evident.

Considerable doubt was cast also upon the practice of sampling through tubing even when bends, horizontal airflows and excessive lengths of tubing were avoided. The convenience of using tubes to obtain samples from some sites is often of paramount consideration but the validity of results with this method should be suspect. Particularly is this so, when only a few bacteria-carrying particles per 100 cubic feet of air sampled may bear a specific pathogen, say, Staph. aureus. Formulae have been defined to allow estimation of losses in tubing (Bourdillon, Lidvall & Lovelock 1948) but it seems desirable to avoid the use of tubes whenever possible and to sample directly from the atmosphere. It would have been interesting and, perhaps, informative to test a piece of smooth-bore, electrically-earthed rubber tubing with similar dimensions to those of the glass tubing used in my tests.

This might have clarified the potential role of electrostatic attraction as a cause of deposition of particles in tubing. However, suitable material could not be obtained.

Finally, these experiments demonstrate the major part played by airflows and structural openings in the transfer of airborne bacteria. (Wells 1955; Lidwell 1961; Bond & Michaelsen 1964; Wolf, Harris & Hall 1961; Ma 1965) Airflows may be enhanced or impeded by the absence or presence of physical obstructions, such as doors. Often, a controlled flow of air is required to ensure that aerial contaminants are transferred from one area to another, as is the case in operating theatre suites. (Lidwell & Bowers 1962) But the success of doors in reducing airflows will be assured only if they remain closed to all but the most essential traffic and this is a matter of human discipline. (Bowers & Crew 1960; Kimmonth et al. 1958)

### 3.2. Ultraviolet Irradiation.

#### 1: Purpose.

This series of experiments was conducted to determine if there was a useful relationship between a photometric and a bacteriological method for estimating the output of ultraviolet (U-V) bactericidal irradiation in the 2537 Å wavelength from commercially available U-V tubes.

#### 2: Materials and Methods.

Twenty U-V tubes of standard, commercial pattern were borrowed from various sources in a number of hospitals. The emission of light at 2537 Å was measured on a photometer

constructed specially for the purpose. This meter was calibrated in relation to a new, unused tube obtained directly from the manufacturer and chosen arbitrarily as exhibiting 100% physical efficiency of output. All photometric readings were taken with the meter placed exactly 50cms. distant from the centre-point of the tube under test.

Bacteriological tests were performed using an overnight broth culture of Staph. aureus in a concentration of approximately  $1 \times 10^5$  organisms per ml.. Viable counts were done by pipetting off five volumes of 0.1 ml. suspension as required and distributing one volume to each of five blood-agar plates. The drops were spread over the agar surface with sterile spreaders before the plates were incubated overnight at 37°C to yield colonies for counting. On a laboratory bench, a gantry was constructed of "Dexion" angle-metal so that the tubes were located during test at a fixed distance of 50cms. from the bacterial suspension when it was exposed in a watchglass exactly below the centre point of the tube. Electric power for the tubes was supplied through a "Variac" transformer which maintained a constant voltage of 240 to ensure that the emission of 2537 Å light did not vary during the duration of the experiment. For similar reasons, it was necessary to "warm up" the tubes before actual use. The type of each tube determined the length of time for this warming-up because double-walled U-V tubes took longer to achieve maximum output than did single-walled tubes. The former were lit for an hour before the test but the latter required only 10 minutes' operation.

### 3: Experimental Procedure.

Thirty Blood-agar were prepared for each test and were

dried in the incubator for two hours before use. The U-V tube for testing was suitably "warmed up". Immediately before the experiment started, the bacterial suspension was diluted 1:100 with saline and 5ml. were placed in a watch-glass which was covered with a black cloth to exclude light rays. 0.5ml. were withdrawn by pipette and plated out as the unirradiated, control counts. The shrouded watchglass was placed in the correct position under the U-V tube and the cloth removed for exactly 12 seconds. The cloth was replaced and the watchglass lifted aside to permit removal of a further 0.5ml. of suspension for plating out. Care was taken to prevent ordinary light reaching the suspension lest photo-reactivation of irradiated cells resulted. The shrouded watchglass was positioned under the tube again and the procedures repeated until a total of 60 seconds' irradiation was completed. Each tube was assessed photometrically both before and after its bacteriological test.

#### 4: Results.

The colony counts obtained for irradiation from each tube are tabulated along with the averages of the 5 counts made for each period of irradiation. (Tables 26-32) Originally, the mean values were plotted on semi-log graph paper on the basis of log. concentration against arithmetic time. The best-fitting straight line, as judged by eye, was drawn through each set of graph points and the slope of each line measured directly as a  $\log$  decline. Tracings of combined sets of these exponential decay-curves are appended. (Figs 9 & 10) The values for these slopes were used to rank each tube in relation to the others. A similar ranking method was applied to the values for physical

efficiency. Pairs of rankings were compared and the difference for each determined. Probabilities were calculated for the frequency of occurrence of various differences in ranking to estimate the comparability of the two experimental methods. (Table 33) Subsequently, I had the opportunity to have the counts processed by an electronic computer. This calculated the slope of the straight line which mathematically, as  $\log_e$  values, best fitted each set of observed counts. (Table 34) I plotted these slope values as a log-log relationship against the values for physical efficiency. (Fig. 11)

### 5: Discussion.

Ultraviolet light is used for its bactericidal effects in several locations in hospitals among them being the special inoculation cabinets in pathological laboratories. Other places for use are in cabinets for the protection of hand wounds during surgical-dressing procedures, (Sussman, Barnes & Lenihan 1961) in operating theatres and wards for disinfection of air, (Wells 1955; Laurell & Ronge 1935; Stratford 1963b; Howard, R.B. et al. 1964) and in water containers where sterilisation of the fluid is desired. (Stratford 1963a; Fredette 1963) The ability of U-V rays to reduce the concentration of aerial bacteria has been amply demonstrated (Rentschler, Nagy & Neuremsoff 1941; Rentschler & Nagy 1942) but it is of practical importance that the output of the bactericidal rays does decline without any commensurate reduction in the emission of the visible light. Use of a substance which fluoresces only in the rays of the germicidal wavelength will indicate this failure of output and beryllium powder has been recommended for this purpose. (Stratford 1963a) Such indicators do not give a quantitative measure of



the efficiency of the U-V tubes and therefore detect only gross inefficiency of irradiation.

Bacteriological testing as a routine is not a practical proposition whereas photometric testing is both quick, simple and accurate if the method is known to relate to bactericidal efficiency. I am not conversant with the physics of the photometric measuring equipment and must accept the validity of the methods employed for it. However, within the limits of error for bacteriological techniques, (Meynell & Meynell 1965) particularly as applicable to viable counts, I consider that the results of photometric measurements do compare satisfactorily with those obtained bacteriologically. On this basis, I suggest that certain criteria be established for describing the efficiency of U-V tubes for hospital use. If the bactericidal efficiency of a tube operating at full output for 60 seconds and achieving a reduction in surface concentration from 300 colonies to 1 colony is rated as "satisfactory", the equivalent range of physical efficiencies is 70-100%. If a reduction to between 2 and 10 colonies is deemed "doubtful" the appropriate equivalent is 50-70%. The description "unsatisfactory" would apply to tubes of less than 50% efficiency.

### 8.3. Applied Air Hygiene.

#### 1: Purpose.

The publication of so many reports detailing features of airborne infection in hospital has focussed the attention of many members of hospital staff on problems, both real and imaginary, associated with their own departments. It was

not surprising, therefore, to find at times that the hospital bacteriologist was invited to investigate particular situations or activities and to advise upon methods to deal with any actual or potential bacterial risks.

## 2: Materials and Methods.

Air sampling was performed with a Casella slit-sampler whenever this was possible but on occasions it was more convenient to use only settle-plate sampling despite the lack of relationship between the two methods.. The duration of exposure was chosen for each investigation in respect of the nature of the sampling site and of the information sought. If short-term variations associated with particular events were under examination, the slit-sampler was used to sample volumes of air for periods of 5 minutes, at intervals not exceeding 5 minutes. But if average concentrations of particles settling during prolonged periods of time were the measurements of choice, then settle-plates were employed for sampling.

Samples were usually taken onto blood-agar plates which were incubated aerobically overnight at 37°C. On a few occasions, neomycin-serum-agar plates were used to sample for Cl. volchii and these were incubated anaerobically. All colonies were counted to determine the total bacterial concentrations sampled. Slit-sample counts were expressed as the number of bacteria-carrying particles per cubic foot of air sampled. Settle-plate counts were converted to numbers of bacteria-carrying particles settling per square foot of surface area per hour. Any potential pathogens were identified by routine bacteriological methods and were differentiated on the basis of antibiotic sensitivity tests

and, for Staph. aureus, bacteriophage typing.

If airflows were thought to be important, as in an operating theatre suite, smoke tubes were used to detect the direction of these flows at doorways. Details were recorded of activities observed to be in progress at different times during sampling so that variations in aerial concentrations of bacteria could be related to specific environmental events. Whenever possible, pathogens in the human population of the area under investigation, were also identified and compared with the aerial isolates to discover if strains in air and in humans were related.

### 3: Situations investigated.

Eight different situations were examined. Four investigations applied to operating theatres or other surgical departments, two related to disposal facilities in wards and the remaining two dealt with staphylococcal infection in wards.

The first air hygiene study was undertaken in a general surgical theatre as part of an investigation of conditions in the theatre. The study occupied two weeks and was designed to demonstrate to the staff the advantage to be gained with input ventilation. During the first week, the existing ventilating practice of extraction was continued but during the second week both input and extract fans were in operation. Settle-plates were exposed at four sites in the theatre suite of which two were in the theatre, one being near the operating table and the other well removed from it. The duration of exposure was related to two periods of the day to try to reveal effects for both busy and quiet conditions. Smoke tests were performed but only total bacterial counts

were made. Two other theatres were examined - a second general surgical theatre and an orthopaedic theatre. Both surgical units had been involved in episodes of post-operative sepsis thus the purpose of the studies was to find if deficiencies in theatre practices could have contributed to the incidence of infection. Slit-sampling was performed in the theatre itself and in the anteroom of both suites in order to obtain counts of all bacteria and of any individual pathogens, particularly of Staph. aureus. The general surgical theatre was provided with extract ventilation only, but the orthopaedic unit was supplied with filtered air under slight positive-pressure.

The studies of disposal facilities in wards were undertaken to try to compare bacteriologically the conditions associated with a simple design of disposal chute and those existing when a disposal system, including its chute, was specially designed to minimise spread of infection. The simple system depended upon a plain vertical shaft with access hatchways into the dirty utility rooms of the wards on each floor it passed through. The chute was used only for linen in bags and the access openings were closed off by hinged doors. The utility rooms, both clean and dirty, were not specially ventilated nor were the other ward areas. In contrast, the special system included extract ventilation of the dirty utility room and input ventilation of the adjoining treatment room. The access openings to both the linen and the refuse chute were fitted with double-ended loading bins which effectively formed air-locks. Bagged materials could not be discharged into either chute unless the outer lid of the bin was securely closed. All soiled or fouled linen was decontaminated and then mechanically sluiced before being

bagged for discharge. Settle-plate samples were obtained for disposal areas in a theatre suite, in a ward with access to the chute, in a ward without access to the chute, and in the basement area where the chutes discharged. Samples from several sites in each area were taken simultaneously to differentiate between aerial contamination arising in the proximity of the chute and that arising elsewhere in the area. The sampling periods were chosen to reflect the periods of maximum, minimum and average activity for each area. Records were made of the usage of each chute during each sampling period and also of the isolations of specific pathogens either from the air samples or from the patients. Slit-sampling was undertaken for only short periods in each area during disposal activities.

Ventilation of a cardiac catheterisation room was studied to determine how best to use existing facilities. Either input or extract flow could be obtained with a ventilation plant equipped with filters on its outside ducts. Separate louvred ducts also extended to the outside atmosphere of a busy street at pavement level. Input ventilation was found to produce intolerably warm and stuffy conditions in the room during occupation but members of the staff were reluctant to use the room if only extract ventilation was available. Slit-sampling, and a general examination of the facilities, was undertaken to assess the difficulties.

The two investigations of airborne staphylococcal infection were supplementary to the work of colleagues who were involved in efforts to control outbreaks of such infection in a dermatological and in an orthopaedic unit. Slit-samples were taken in the centre of each male and female ward as well

as in the dermatology treatment room and in an orthopaedic isolation ward. Total bacterial counts as well as counts of Staph. aureus were obtained and all staphylococci which were coagulase positive were phage-typed.

#### 4: Results.

The settle-plate counts for the first study are tabulated in the Appendix. (Table 35) Along with them, are the average counts calculated for the different sites during the different periods of a day. As was expected, the counts for each area varied considerably but those for the corridor and the vestibule were greater than those obtained inside the theatre. Extract ventilation induced flows of air from the hospital into the theatre but this was reversed when the input fans were in operation. Extract ventilation could not be abolished because its main use was to remove excess heat generated in an open steriliser bay on one side of the theatre. Input and extract ventilation did reduce the concentration of aerial contaminants quite markedly except in the vestibule. This site was the focal point for all the activity in the theatre suite. Traffic for any part of the suite had to pass there and probably this accounts for the apparent failure to show improvement. Observations of the traffic suggested that an unduly high frequency of movements to and from the theatre did little to contribute to cleaner environmental conditions.

Undue activity, coupled with a noticeable reluctance on the part of staff to keep doors closed, was a feature of the studies of the other surgical units. The general surgical theatre which had only extract ventilation frequently had an unobstructed flow of air direct from the hospital. Again, one of the complaints was that hot air from the steriliser

room made conditions unbearable in the theatre unless a good flow of cool air was provided. Regrettably, only extract ventilation was available to provide any kind of flow. The orthopaedic theatre did have input ventilation which produced an airflow predominantly from the theatre towards the hospital. The pressurisation was such that counterflows were readily induced when door openings were unobstructed. The major feature of the observations was the frequency with which ten or more persons were present in the theatre during operations. This did not seem to be a peculiarity of the operation which was in progress when slit-sampling was performed. In addition, the ante-room was often used as a minor theatre although proper facilities did not exist there. Overcrowding was common also in the cardiac catheterisation room although the restricted dimensions of the area were not conducive to efficient function. Airflows were obtained in accordance with the type of mechanical process of ventilation chosen - in-flows with extract ventilation and out-flows with pressurised ventilation. Although these were the predominant flows, the existence of the separate ducts to atmosphere allowed considerable variation depending upon the prevailing air currents in the outside air. In some instances, the external pressure exceeded that produced by the ventilation plant and complete reversal of the flows of air resulted momentarily. The slit-sample counts are recorded graphically for each theatre (Figs. 12 & 13) and for the catheterisation room, (Fig. 16, top) in the Appendix. Relevant details of events observed to relate to changes in aerial concentrations and of the numbers and identity of pathogenic isolates are recorded on the diagram also.

The results for the investigation of the two chute systems are recorded in two parts. The settle-plate counts are tabulated (Tables 36 & 37) as too are the average values relating to each site in each area and for each period of time. (Tables 38 & 39) The slit-sampling counts are displayed graphically (Figs. 14 & 15) along with annotations of the related events and bacterial isolations. It was not possible to test directly the extent of bacterial transfer through the two different types of chutes. Smoke tests did demonstrate clearly that very large volumes of air passed upwards and outwards in the simple chute so much so that the topmost doorway afforded almost continuous discharge of air into the top-floor ward. The greater part of this air seemed to originate in the ground-floor ward which accommodated infants. Many fouled napkins were handled for bagging close to the lower access hatchway and possibly faecal bacteria were dispersed upwards to other dirty utility rooms. (D.U.R.) In this context, it may be of significance that coliforms and Cl. welchii were isolated during air sampling in this hospital more often than was Staph. aureus. Only minimal transfer of air through the special chute could be detected. The use of extract ventilation for the dirty utility areas of this other system ensured a constant flow of air from the ward and from the treatment room into the disposal area. Both hospitals had hatchways linking the adjoining treatment and disposal rooms. It was observed that the prevailing airflow was from "clean" to "dirty" with the extract ventilated arrangement but the reverse of this in the naturally ventilated hospital.

Extract ventilation was provided for the dermatology treatment room but this did not afford much benefit for cleanliness of air when compared with ward standards. Both



ward units were of the old-fashioned, naturally ventilated, "Nightingale" type with 24 beds arranged round the periphery of a large rectangular room. Domestic facilities and the duty room were situated at one end and sanitary accommodation at the other. It was impossible to relate observed bacterial concentrations to anything more specific than general patterns of ward activity. The results of slit-sampling are recorded in graphic form and details of these patterns are included. (Figs. 16, bottom, & 17) Total bacterial counts are drawn in but actual numbers of colonies of particular phage-types of Staph. aureus are noted. Staphylococcal strains isolated during the epidemic outbreaks in the dermatological female ward and in the orthopaedic male ward before cleaning were readily related to patients infected with the same strains. Isolations during non-epidemic periods in the same wards or other wards of the same units were often apparently unrelated to any detectable source.

### 5: Discussion.

Certain general observations can be made regarding the results recorded. Both settle-plate sampling (Table 35) and slit-sampling (Figs. 13 & 16, top) show the effect of input ventilation as a means to reducing concentrations of airborne bacteria. Equally, they both revealed increases in total bacteria in air or sedimenting from air during or after human activity - transfer of patients to and from the operating table, (Figs. 12, 13 & 16, top) washing of floors, (Fig. 14) handling of loose linen, (Fig. 15) bedmaking (Fig. 17) and dressing and undressing. (Fig. 16) But the appearance of pathogens in air even during epidemic conditions was not directly related in time or in numbers to the presence of

general bacteria. (Figs. 16, bottom & 17) Probably this difference reflects the different sites of origin of the two groups of contaminants - Staph. aureus derived directly from humans and general bacteria present mainly on dust particles. During epidemics, the types of pathogens found in air usually correspond to the types known to be affecting the patients. (Fig. 16 - female ward, Fig. 16 - male ward and isolation ward) At other times, when sources of aerial pathogens could not be found, I would suggest that these stray isolates are dust-borne parasites existing in the environmental part of the "life cycle" of hospital infection. (Fig. 12, 13 & 17) In the disposal areas, handling of the contaminated linen gave rise to gross dispersal of contaminants. (Tables 37 & 38) This was particularly noticeable where procedures to minimise handling and dispersal were not enforced. In this respect, it is significant that the sorting of this linen was performed close to the sampling site distant from the simple chute. The degree of contamination at this site provides ample evidence of the unhygienic nature of the procedures. More disturbing, however, was the observation that this dispersal arose close to the hatchway connecting the ward treatment room to the dirty utility area and that the airflow through this opening probably conveyed large numbers of organisms into what was supposed to be a clean area. Very considerable dispersal was found also in the dermatology ward apparently as a normal occurrence although it was well in excess of the dispersal associated with the orthopaedic ward.

These findings are in agreement with the observations of other investigators whose reports have been referred to before in this thesis. The over-riding impression gained is that, despite the publication of these reports, relatively little

has been done, let alone accomplished, to benefit from them either by physical improvements or by better organisation of human activities. Certainly, of the hospitals I visited during my investigations, only one had been built so recently as to provide the opportunity to attempt planned control of infection hazards. In this planning, air hygiene was taken as an integral part as is evidenced by the provision of input ventilation for the ward treatment rooms, extract ventilation for the adjacent dirty utility rooms, and special air-lock loading bins for the linen and the refuse chutes. Other instances of forethought based upon existing knowledge of hospital infection is the existence of ward isolation-rooms, the disinfection and mechanical sluicing of fouled and infected linen before it is handled loose and bagged, and the use of plastic lining-bags for all waste receptacles to eliminate loose handling of refuse.

The microscopic differences detected between these disposal facilities and the ones available in the hospital with the simple chute only confirmed what could be discerned macroscopically. Reliance upon natural ventilation for maintenance of directional airflow, absence of forethought in planning wards for efficient operation, and persistent application of unhygienic methods such as manual sorting of fouled and infected linen all contributed to the existence of an untidy and bacteriologically unclean establishment. Agreed - this hospital was planned and built before the full appreciation of the concepts of control of hospital infection had begun to have effect. Patent proof of this was found in the provision of positive-pressure ventilation independently for each of a pair of operating theatres but the omission of any suitable outflow in the intervening dirty utility room.

Consequently, when one theatre's ventilation system was not in use, the positive pressure in the other forced contaminated air from the disposal area into the unventilated theatre. Furthermore, independent access to the disposal area had not been provided and all staff entering or leaving this area had to pass through one or other of the operating theatres.

With this in mind, possibly one should not be too critical of the hygienic failings of other hospitals built before the turn of this century. Little can be done to improve the structural layout of many of these old buildings either in operating theatres or wards although the modern practice is to divide up the ward space in a "Nightingale" ward into a series of open cubicles. However, the limitations on change in structure are often used as excuses to postpone or reject a re-organisation of the human activities within the hospital. Such elementary changes as the correct use of existing ventilating equipment, the closing of doors when these are provided and the provision of doors when these are absent at doorways, were relatively easily accomplished. But the essential changes required are in the attitudes of the medical, nursing and domestic staffs who are directly responsible for the care and health of the patients in their wards and theatres.

### 3.4. General Discussion.

Although the personal investigations described have each been undertaken and reported separately, they all bear in some way upon the one topic of "hospital infection". Air hygiene is only one facet of this peculiarly institutional problem, as I have attempted to show. Undoubtedly, airborne infection has received much attention during the past 25 years and particularly during the last decade. Despite this, opinions differ widely

regarding its importance. These differences of opinion among medical bacteriologists have served to confuse the point at issue, not just for themselves but more so for their clinical colleagues. The problem is not whether one route for spread of infection is more important over all than another but rather when and where is one route more advantageous to pathogens compared with another. Herein lies the crux of the matter because accurate assessment of the conditions existing on one route in one particular department of one hospital presupposes that there is available accurate and up-to-date information on the conditions existing for all routes in that situation. The practical implications of this statement are such that the most experienced hospital microbiologist would draw back in horror if asked to attempt such a task.

My investigations indicate a few of the many diverse problems of air hygiene which may require to be considered by a hospital bacteriologist. Even in the absence of a slit-sampler, he can attempt several simple investigations if only to provide practical demonstrations of the benefits to be derived from application of the principles he advocates verbally. The time and effort absorbed by even elementary investigations of this kind are often all that a busy laboratory worker can spare. More ambitious investigations require proper facilities and staff which few hospitals can afford individually. The establishment of an experienced team to deal with problems of hospital infection would be a feasible proposition for a hospital group or a regional board. However, to be of most value and effect, such a team would have to consist predominantly of engineers who were thoroughly conversant with the technicalities of the problems facing them.

### 8.5. Conclusions.

Aerial transfer of pathogenic bacteria is an established fact. Its frequency and magnitude varies greatly from place to place and from time to time. The risk of infection with which it is associated has not been defined as yet but measures which reduce concentrations and which direct the flows of transfer are to be recommended generally. Undue emphasis should not be placed upon individual aspects of airborne infection to the exclusion of others because seldom does such exclusion apply in reality. Interactions are the rule, not the exception, both in air hygiene and in the wider sphere to which it belongs - control of infection in hospital. However, the very variety of the problems requiring solution and the need to devote adequate time and resources to their study indicates that future investigation of air hygiene in hospital is likely to become the province of experienced technicians rather than of medical bacteriologists.

## 4 : APPENDIX

Arranged in order of -

Tables : Pages T.1 - 39

Figures : Pages F.1 - 17

References : Pages R.1 - 20

and Acknowledgements.

Table 1. Counts of airborne bacteria-carrying particles per 0.5 cubic foot of air sampled, sampler W.

Time Mins	Small ward - run 1		Small ward - run 2	
	Count	Range	Count	Range
0	5	4-6	2	1-3
1	84	82-86	62	60-64
2	217	214-220	202	199-205
3	411	407-415	384	380-388
4	313	309-317	328	324-332
5	261	257-265	246	242-250
6	215	212-218	224	221-227
7	196	193-199	221	218-224
8	171	168-174	191	188-194
9	169	166-172	178	175-181
10	160	157-163	155	152-158
11	149	146-152	165	162-168
12	142	139-145	152	149-155
13	145	142-148	150	147-153
14	133	130-136	137	134-140
15	128	125-131	131	128-134
16	124	121-127	130	127-133
17	116	113-119	122	119-125
18	109	106-112	113	110-116
19	107	104-110	108	105-111
20	99	96-102	91	88-94
21	87	85-89	90	88-92
22	85	83-87	82	80-84
23	86	84-88	75	73-77
24	72	70-74	69	67-71
25	70	68-72	61	59-63
26	68	66-70	51	49-53
27	61	59-63	53	51-55
28	55	53-57	45	43-47
29	49	47-51	42	40-44



Table 2. Counts of airborne bacteria-carrying particles per 0.5 cubic feet of air sampled, sampler W.

Time Mins	Small ward - run 3		Duty room - run 1	
	Count	Range	Count	Range
0	3	2-4	15	14-16
1	77	75-79	521	516-526
2	189	186-192	1091	1084-1098
3	351	347-355	1120	1113-1127
4	421	416-426	937	931-943
5	344	340-348	739	724-756
6	310	306-314	635	630-640
7	291	287-295	576	571-581
8	268	264-292	498	493-503
9	251	247-255	438	433-443
10	246	243-250	394	390-398
11	237	234-241	373	369-377
12	245	241-249	335	331-339
13	222	218-226	307	303-311
14	197	194-200	271	267-275
15	183	180-186	215	212-218
16	177	174-180	233	229-237
17	158	155-161	183	180-187
18	161	158-164	166	163-169
19	152	149-155	147	144-151
20	123	120-126	129	126-132
21	111	108-114	135	132-138
22	100	97-103	122	119-125
23	109	106-112	111	108-114
24	102	99-105	97	94-100
25	93	90-96	82	80-84
26	90	88-92	72	70-74
27	83	81-85	59	58-62
28	73	71-75	75	73-77
29	71	69-73	67	65-69

Table 3. Counts of airborne bacteria-carrying particles per 0.5 cubic foot of air sampled, sampler W.

Time Mins	Duty room - run 2		Duty room - run 3	
	Count	Range	Count	Range
0	10	0-11	9	0-10
1	887	881-893	206	203-209
2	1202	1205-1209	757	751-763
3	1142	1135-1149	1015	1008-1022
4	766	762-774	819	813-825
5	723	717-729	783	776-789
6	580	581-591	721	715-727
7	557	552-562	679	674-684
8	536	531-541	606	601-611
9	424	419-429	548	543-553
10	400	396-404	505	500-510
11	363	349-357	480	484-504
12	334	330-338	427	422-432
13	301	297-305	407	403-411
14	284	280-288	366	362-370
15	259	255-263	343	338-347
16	255	251-259	291	287-295
17	223	219-227	277	273-281
18	217	214-220	225	221-229
19	179	176-182	213	210-216
20	182	179-185	199	196-202
21	177	174-180	201	198-204
22	163	160-166	184	181-187
23	133	130-136	160	157-163
24	134	131-137	145	142-148
25	128	125-131	133	130-136
26	117	114-120	121	118-124
27	103	100-106	118	115-121
28	95	92-98	106	103-109
29	93	90-96	100	97-103

Table 4. Counts of airborne bacteria-carrying particles per 0.5 cubic feet of air sampled, sampler W.

Time mins	Large ward - run 1		Large ward - run 2	
	Count	Range	Count	Range
0	1	1	1	1
1	3	2-4	10	2-11
2	52	50-54	74	72-76
3	132	179-133	215	212-218
4	263	259-267	336	332-340
5	345	340-350	512	507-517
6	399	395-403	411	407-415
7	377	373-381	301	297-305
8	364	360-368	276	272-280
9	313	309-317	262	258-266
10	297	293-301	250	246-254
11	276	272-280	233	229-237
12	246	242-250	209	206-212
13	213	210-216	197	194-200
14	200	206-212	187	184-190
15	202	199-205	173	170-176
16	181	178-184	172	169-175
17	178	175-181	166	163-169
18	162	179-185	162	159-165
19	165	162-168	155	152-158
20	163	160-166	143	145-151
21	156	153-159	143	140-146
22	139	136-142	139	136-142
23	128	125-131	133	130-136
24	125	122-128	127	124-130
25	113	110-116	121	118-124
26	109	106-112	115	112-118
27	106	103-109	106	103-109
28	100	97-103	97	94-100
29	98	95-101	86	84-88

Table 5. Counts of airborne bacteria-carrying particles per 0.5 cubic feet of air sampled, sampler W.

Time Mins	Large vord - run 3		Large vord - run 4	
	Count	Range	Count	Range
0	9	2-4	1	1
1	11	10-12	5	4-6
2	64	62-66	59	57-61
3	184	181-187	204	201-207
4	419	416-422	293	288-297
5	601	596-606	496	491-501
6	540	535-545	455	450-460
7	321	317-325	383	379-387
8	263	270-267	319	315-323
9	255	251-259	244	240-248
10	230	226-234	237	233-241
11	191	188-194	219	216-222
12	179	176-182	182	179-185
13	171	169-174	169	166-172
14	160	157-163	161	158-164
15	152	149-155	154	151-157
16	143	140-146	142	139-145
17	130	127-133	136	133-139
18	129	126-132	127	124-130
19	117	114-120	121	116-124
20	112	109-115	115	112-118
21	107	104-110	110	107-113
22	105	102-108	101	98-104
23	92	89-95	94	91-97
24	90	87-93	92	89-95
25	91	88-94	89	87-91
26	88	86-90	80	78-82
27	84	82-86	79	77-81
28	75	73-77	77	75-79
29	73	71-75	71	69-73

Table 6. Counts of airborne bacteria-carrying particles per 0.5 cubic feet of air sampled, sampler R.

Time Mins	Small ward - run 1		Small ward - run 2	
	Count	Range	Count	Range
0	13	12-14	17	16-18
1	178	175-181	187	184-190
2	305	301-309	330	326-334
3	285	281-289	312	308-316
4	260	256-264	263	259-267
5	225	221-229	231	227-235
6	190	187-193	204	201-207
7	181	178-184	193	190-196
8	162	159-165	173	170-176
9	159	156-162	157	154-160
10	144	141-147	149	146-152
11	134	131-137	130	127-133
12	125	122-128	122	119-125
13	114	111-117	117	114-120
14	107	104-110	101	98-104
15	97	94-100	95	92-98
16	83	81-85	79	77-81
17	72	70-74	70	68-72
18	65	63-67	61	59-63
19	57	55-59	55	53-57
20	50	48-52	46	44-48
21	46	44-48	41	39-43
22	41	39-43	36	34-38
23	37	35-39	33	31-35
24	34	32-36	29	27-31
25	31	29-33	27	25-29
26	25	23-27	24	22-26
27	23	21-25	23	21-25
28	18	17-19	20	19-21
29	16	15-17	15	14-16

Table 7. Counts of airborne bacteria-carrying particles per 0.5 cubic feet of air sampled, sampler H.

Time Mins	Small vord - run 3		Small vord - run 4	
	Count	Range	Count	Range
0	15	14-16	11	10-12
1	195	192-198	167	164-170
2	281	277-285	272	268-276
3	305	301-309	309	305-313
4	270	266-274	267	263-271
5	242	238-246	209	206-212
6	192	189-195	190	177-183
7	185	182-188	172	169-175
8	164	161-167	161	158-164
9	146	143-149	148	145-151
10	137	134-140	135	132-138
11	125	123-129	121	118-124
12	118	115-121	111	108-114
13	110	107-113	105	102-108
14	99	96-102	96	93-99
15	89	87-91	82	80-84
16	77	75-79	71	69-73
17	69	67-71	64	62-66
18	60	58-62	58	56-60
19	57	55-59	53	51-55
20	51	48-53	47	45-49
21	43	41-45	40	38-42
22	38	36-40	32	30-34
23	30	28-32	25	23-27
24	26	24-28	22	20-24
25	24	22-26	18	17-19
26	20	19-21	16	15-17
27	18	17-19	15	14-16
28	14	13-15	13	12-14
29	12	11-13	10	9-11

Table 3. Counts of airborne bacteria-carrying particles per 0.5 cubic feet of air sampled, samplers W & R.

Time Mins	Small ward Run 1		Small Ward Run 2		Small Ward Run 3	
	W	R	W	R	W	R
0	1	3	3	4	1	2
1	79	86	64	71	66	69
2	318	337	243	255	199	211
3	269	282	337	390	357	363
4	243	251	338	344	285	292
5	235	239	288	301	249	256
6	219	223	270	277	236	240
7	205	203	230	243	230	226
8	173	186	211	225	210	204
9	155	160	194	193	188	193
10	141	149	181	180	173	182
11	132	138	176	173	170	179
12	120	122	159	161	163	166
13	109	114	133	142	151	159
14	104	103	126	134	144	151
15	93	97	121	127	137	140
16	90	91	114	119	133	133
17	81	86	103	110	120	122
18	76	73	97	103	107	114
19	72	72	92	99	100	105
20	66	69	86	90	94	97
21	62	65	83	87	89	92
22	59	60	79	81	83	85
23	56	53	75	74	75	80
24	50	52	70	68	70	76
25	45	44	65	62	66	71
26	42	40	57	56	62	67
27	38	37	48	50	53	63
28	34	35	40	43	55	53
29	29	31	34	39	50	52

Table 9. Counts of airborne bacteria-carrying particles per 0.5 cubic feet of air sampled, samplers R & W.

Time mins	Small ward Run 2		Small ward Run 5		Small ward Run 6	
	R	W	R	W	R	W
0	7	5	3	2	3	1
1	98	96	60	56	79	74
2	355	340	237	231	270	268
3	476	465	346	336	359	347
4	340	336	255	242	251	240
5	294	287	231	225	212	210
6	253	248	213	205	193	189
7	221	213	192	190	174	175
8	203	204	174	170	157	155
9	196	192	162	156	149	139
10	179	172	155	143	133	136
11	162	160	149	143	123	116
12	155	149	141	137	116	111
13	150	146	135	130	110	103
14	144	140	129	126	107	100
15	138	135	124	121	99	95
16	133	129	120	117	91	86
17	126	122	113	113	84	81
18	120	114	108	107	78	75
19	113	110	101	102	68	66
20	109	108	96	93	62	60
21	102	100	91	88	59	58
22	96	94	83	80	53	53
23	84	86	78	73	47	43
24	80	81	73	70	41	36
25	75	73	69	64	38	33
26	71	68	66	61	34	30
27	66	62	60	56	29	26
28	69	63	55	54	25	23
29	52	50	49	47	22	18



Table 10. Differences between colony-counts for air-samples taken simultaneously by samplers W and R.

Time Mins	Small ward Run 1		Small ward Run 2		Small ward Run 3	
	W ± R	W ± R ≥ Range	W ± R	W ± R ≥ Range	W ± R	W ± R ≥ Range
0	2	=	1	=	1	=
1	7	+R	7	+R	3	=
2	10	+R	12	+R	12	+R
3	13	+R	9	+R	11	+R
4	8	=	6	=	7	=
5	4	=	13	+R	9	+R
6	9	+R	7	=	4	=
7	3	=	13	+R	4	=
8	8	+R	14	+R	6	=
9	6	=	4	=	6	=
10	6	+R	1	=	4	=
11	6	=	3	=	9	+R
12	2	=	2	=	6	=
13	6	=	4	=	8	+R
14	1	=	8	+R	7	+R
15	4	=	6	=	3	=
16	1	=	5	=	0	=
17	6	+R	2	=	2	=
18	2	=	6	=	7	+R
19	0	=	7	+R	6	=
20	3	=	4	=	3	=
21	3	=	4	=	3	=
22	1	=	2	=	2	=
23	2	=	1	=	6	+R
24	2	=	2	=	6	+R
25	1	=	3	=	6	+R
26	2	=	1	=	6	+R
27	1	=	2	=	5	+R
28	1	=	3	=	3	=
29	2	=	6	+R	2	=

Table 11. Differences between colony-counts for air-samples taken simultaneously by samplers R and W.

Time Mins	Small ward Run 4		Small ward Run 5		Small ward Run 6	
	R ± W	R ± W > Range	R ± W	R ± W > Range	R ± W	R ± W > Range
0	2	=	1	=	2	=
1	2	=	4	=	5	+R
2	15	+R	0	=	3	=
3	11	+R	10	+R	12	+R
4	4	=	13	+R	11	+R
5	7	=	3	=	2	=
6	5	=	3	+R	4	=
7	0	+R	2	=	1	=
8	1	=	4	=	2	=
9	4	=	0	=	1	=
10	7	+R	7	+R	2	=
11	2	=	0	=	7	+R
12	0	=	4	=	5	=
13	4	=	5	=	7	+R
14	4	=	3	=	7	+R
15	3	=	3	=	4	=
16	4	=	3	=	5	+R
17	4	=	0	=	3	=
18	0	=	1	=	0	=
19	3	=	1	=	2	=
20	1	=	1	=	2	=
21	2	=	3	=	1	=
22	2	=	3	=	0	=
23	2	=	5	+R	4	=
24	1	=	3	=	5	+R
25	2	=	5	+R	5	+R
26	3	=	5	+R	4	=
27	4	=	2	=	3	=
28	1	=	1	=	2	=
29	2	=	2	=	4	=

Table 12. Probability of obtaining comparable air-sample counts with samplers W and R.

Frequency of events:

Run 1 W : R		
R > W	7	
W > R	0	
R = W	23	

Run 2 W : R		
R > W	9	
W > R	0	
R = W	21	

Run 3 W : R		
R > W	12	
W > R	0	
R = W	23	

Run 4 R : W		
R > W	4	
W > R	0	
R = W	23	

Run 5 R : W		
R > W	7	
W > R	0	
R = W	23	

Run 6 R : W		
R > W	0	
W > R	0	
R = W	21	

Probability of ~	by positions		not by position
	R : W	W : R	
R > W	0.31	0.22	0.27
W > R	0.00	0.00	0.00
R = W	0.69	0.78	0.73

Table 13. Counts of airborne bacteria-carrying particles per 0.5 cubic feet of air sampled, samplers W & R

Time Mins	Small ward - run 1		Small ward - run 2	
	W	R/tube	W	R/tube
0	4	1	6	2
1	24	14	21	13
2	119	100	122	107
3	254	284	290	263
4	388	361	264	238
5	293	265	227	201
6	208	184	213	194
7	180	153	209	186
8	162	144	185	172
9	148	121	176	156
10	126	109	162	151
11	117	103	160	144
12	107	88	153	140
13	101	86	146	133
14	96	80	141	129
15	82	71	136	126
16	79	68	131	120
17	72	60	128	114
18	66	54	123	111
19	59	49	119	108
20	55	46	114	102
21	53	43	109	97
22	48	39	100	91
23	44	34	90	81
24	41	33	87	76
25	37	30	83	72
26	34	27	74	65
27	30	26	69	61
28	28	21	63	54
29	22	19	60	52

Table 14. Counts of airborne bacteria-carrying particles per 0.5 cubic feet of air sampled, samplers W & R

Time Mins	Small ward - run 3		Small ward - run 4	
	R	W/tube	R	W/tube
0	7	8	11	5
1	25	12	23	14
2	133	107	145	130
3	225	186	327	301
4	289	266	535	493
5	336	298	398	367
6	307	272	342	321
7	248	221	284	256
8	229	196	258	229
9	216	182	234	202
10	205	177	199	176
11	184	150	175	160
12	161	143	164	148
13	152	135	157	142
14	145	120	144	131
15	128	109	139	128
16	116	100	135	122
17	104	92	124	115
18	92	81	117	106
19	85	74	105	92
20	77	67	98	86
21	72	63	86	79
22	64	54	81	70
23	59	52	71	62
24	51	46	65	57
25	47	40	60	51
26	43	35	52	43
27	35	30	44	38
28	31	27	39	34
29	26	21	33	30

Table 15. Differences between colony-counts for air-samples taken simultaneously by samplers W & R/tube.

Time Mins	Small word - run 1		Small word - run 2	
	W $\pm$ R/tube	W $\pm$ R/tube $\geq$ Range	W $\pm$ R/tube	W $\pm$ R/tube $\geq$ Range
0	3	+W	4	+W
1	10	+W	3	+W
2	10	+W	15	+W
3	20	+W	27	+W
4	27	+W	26	+W
5	28	+W	26	+W
6	24	+W	19	+W
7	27	+W	23	+W
8	18	+W	13	+W
9	27	+W	20	+W
10	15	+W	11	+W
11	14	+W	16	+W
12	10	+W	13	+W
13	15	+W	13	+W
14	16	+W	12	+W
15	11	+W	10	+W
16	16	+W	11	+W
17	12	+W	14	+W
18	12	+W	12	+W
19	10	+W	11	+W
20	9	+W	13	+W
21	10	+W	12	+W
22	9	+W	9	+W
23	10	+W	9	+W
24	8	+W	11	+W
25	7	+W	11	+W
26	7	+W	9	+W
27	4	=	8	+W
28	1	+W	9	+W
29	3	=	8	+W

Table 16. Differences between colony-counts for air-samples taken simultaneously by samplers R & W/tube.

Time Mins	Small ward - run 3		Small ward - run 4	
	R $\pm$ W/tube	R $\pm$ W/tube > Range	R $\pm$ W/tube	R $\pm$ W/tube > Range
0	4	+R	6	+R
1	13	+R	9	+R
2	26	+R	15	+R
3	39	+R	26	+R
4	23	+R	42	+R
5	40	+R	31	+R
6	35	+R	21	+R
7	27	+R	28	+R
8	332	+R	29	+R
9	34	+R	32	+R
10	28	+R	23	+R
11	34	+R	15	+R
12	18	+R	16	+R
13	17	+R	15	+R
14	26	+R	13	+R
15	10	+R	11	+R
16	16	+R	13	+R
17	12	+R	9	+R
18	11	+R	11	+R
19	11	+R	13	+R
20	10	+R	10	+R
21	9	+R	7	+R
22	10	+R	11	+R
23	7	+R	9	+R
24	5	+R	8	+R
25	7	+R	9	+R
26	8	+R	9	+R
27	5	+R	6	+R
28	4	=	8	+R
29	5	+R	3	=

Table 17. Probability of obtaining comparable air-sample counts with samplers W and R, through tubing.

Frequency of events:

Run 1 W : R/tube	
R > W	0
W > R	28
R = W	2

Run 2 W : R/tube	
R > W	0
W > R	30
R = W	0

Run 3 R : W/tube	
R > W	29
W > R	0
R = W	1

Run 4 R : W/tube	
R > W	29
W > R	0
R = W	1

Probability of -	pairing of machine & tube		tube only
	R/tube : W	R : W/tube	
R > W	0.00	0.97	0.485
W > R	0.97	0.00	0.485
R = W	0.03	0.03	0.03



Table 18. Numbers of colonies counted per plate.

Plate	Operator A			Operator B			Operator C		
	1	2	3	1	2	3	1	2	3
1	583	576	591	582	588	590	593	587	591
2	464	458	446	459	450	461	457	462	454
3	376	371	382	380	375	383	377	374	381
4	335	324	330	328	341	333	327	335	330
5	292	284	290	287	285	291	283	289	290
6	234	231	233	235	230	234	236	233	233
7	200	206	195	199	205	201	200	205	201
8	171	164	169	165	160	163	167	165	166
9	152	156	151	150	155	151	157	152	154
10	139	141	139	140	142	137	140	138	141
11	125	127	133	126	132	129	131	128	128
12	118	124	117	120	123	119	116	121	117
13	110	112	106	111	108	109	107	112	108
14	101	102	106	103	104	104	105	101	104
15	98	96	100	98	98	96	100	97	98
16	76	75	71	72	75	74	70	73	71
17	68	63	66	65	63	65	66	63	64
18	54	58	57	55	57	56	53	56	55
19	40	36	35	39	35	38	37	35	37
20	32	28	33	30	31	28	29	32	31
21	20	23	21	20	21	19	20	22	20
22	16	17	15	16	16	15	17	16	15

Table 19. Mean counts per plate.

Plate	Counts	
	Sum	Mean
1	5271	586
2	4106	456
3	3399	378
4	2992	332
5	2591	288
6	2113	235
7	1812	201
8	1490	166
9	1378	153
10	1256	140
11	1159	129
12	1075	120
13	983	109
14	933	104
15	881	98
16	657	73
17	583	65
18	506	56
19	332	37
20	274	30
21	186	21
22	143	16

Table 20. Deviation from mean, per plate.

Plate	Operator A			Operator B			Operator C		
	1	2	3	1	2	3	1	2	3
1	-3	-10	+5	-4	+2	+4	+7	+1	-5
2	+6	-3	-10	+3	-6	+5	+1	+6	-2
3	-2	-7	+4	+2	-3	+5	-1	-4	+3
4	+3	-8	+7	-4	+9	-9	-5	+3	-2
5	+4	-4	+2	-1	-3	+3	-5	+1	+2
6	-1	-4	+3	0	+4	-1	+1	-2	-2
7	-1	+5	-6	-2	+4	0	-1	+4	0
8	+6	-2	+3	-1	-6	-3	+1	-1	0
9	-1	+3	-2	-3	+2	-2	+4	-1	+1
10	-2	+1	-1	0	+2	-3	0	-2	+1
11	-4	-2	+4	-3	+3	0	+2	-1	-1
12	-2	+4	-3	0	+3	-1	-4	+1	-3
13	+1	+3	-3	+2	-1	0	-2	+3	-1
14	-3	-2	+2	+2	0	0	+1	-3	0
15	0	-2	+2	0	0	-2	+2	-1	0
16	+3	+2	-2	-1	+2	+1	-3	0	-2
17	+3	-2	+1	0	-2	0	+1	-2	-1
18	-2	+2	+1	-1	+1	0	+2	0	-1
19	+3	-1	-2	+2	-2	+1	0	-2	0
20	+2	-2	+3	0	+1	-2	-1	+2	+1
21	-1	+2	0	-1	0	-2	-1	+1	-1
22	0	+1	-1	0	0	-1	+1	0	-1

Table 21. Average deviation from mean, per plate.

Plate	Average Deviation	
1	+3.80	-5.50
2	+4.00	-5.25
3	+3.50	-3.40
4	+5.50	-5.00
5	+2.40	-3.25
6	+2.00	-2.00
7	+4.33	-2.50
8	+3.00	-2.00
9	+2.50	-1.80
10	+1.33	-2.00
11	+3.00	-2.20
12	+2.33	-2.00
13	+2.25	-1.75
14	+1.66	-2.00
15	+2.00	-1.66
16	+2.00	-2.00
17	+1.66	-1.75
18	+1.50	-1.33
19	+2.00	-1.75
20	+1.20	-1.86
21	+1.50	-1.20
22	+1.00	-1.00

Table 22. Frequencies of deviation from mean.

Plate	Operator A			Operator B			Operator C			A + B + C		
	+	-	0	+	-	0	+	-	0	+	-	0
1	1	2		2	1		2	1		5	4	
2	1	2		2	1		2	1		5	4	
3	1	2		2	1		1	2		4	5	
4	2	1		1	2		1	2		4	5	
5	2	1		1	2		2	1		5	4	
6	1	2		1	1	1	1	2		3	5	1
7	1	2		1	1	1	1	1	1	3	4	2
8	2	1			3		1	1	1	3	5	1
9	1	2		1	2		2	1		4	5	
10	1	2		1	1	1	1	1	1	3	4	2
11	1	2		1	1	1	1	2		3	5	1
12	1	2		1	1	1	1	3		3	5	1
13	2	1		1	1	1	1	2		4	4	1
14	1	2		1		2	1	1	1	3	3	3
15	3	1	1		1	3	1	1	1	2	3	4
16	2	1		2	1			2	1	4	4	1
17	2	1			1	2	1	2		3	4	2
18	2	1		1	1	1	1	1	1	4	3	2
19	1	2		2	1			1	2	3	4	2
20	2	1		1	1	1	2	1		5	3	1
21	1	1	1		2	1	1	2		2	5	2
22	1	1	1		1	2	1	1	1	2	3	4
Totals	30	33	3	22	27	17	25	31	10	77	91	30

Table 23. Probability of estimating "true" colony-count.

Overall Probability of	Operators			
	A	B	C	A+B+C
- overestimating	0.45	0.33	0.33	0.39
- underestimating	0.50	0.41	0.47	0.46
- "correctly" estimating	0.05	0.26	0.15	0.15
	Regular fast counter	Regular slow counter	Irregular slow counter	

Table 23A. Correction factors for colony-counts.

Range of counts	$\pm$ Deviation from mean
< 20	$\pm 1$
20 - 99	$\pm 2$
99 - 220	$\pm 3$
220 - 420	$\pm 4$
420 - 680	$\pm 5$
680 - 1000	$\pm 6$
> 1000	$\pm 7$

Table 24. Transfer of airborne bacteria-carrying particles within a building, counts per 0.5 cubic feet of air sampled, samplers W and R.

Time Mins	Large Ward W	Small Ward R	Large Ward W	Small Ward R	Duty Room W	Small Ward R
0	0	0	0	2	7	0
1	0	1	0	4	19	1
2	0	2	0	7	31	0
3	1	2	0	9	47	2
4	0	3	2	10	110	1
5	0	5	1	24	300	1
6	1	6	0	27	336	6
7	1	9	0	31	300	10
8	2	13	1	33	233	15
9	1	17	2	36	208	17
10	1	19	0	40	192	22
11	1	24	0	66	172	34
12	2	31	0	104	140	45
13	2	34	2	160	130	43
14	1	46	2	170	113	40
15	3	41	1	155	107	33
16	4	23	1	151	94	33
17	1	26	4	141	92	30
18	2	24	5	134	85	27
19	1	23	5	130	73	25
20	0	25	3	126	93	22
21	2	29	0	119	86	21
22	0	26	2	113	80	20
23	0	22	1	111	73	19
24	0	21	1	107	67	23
25	1	19	3	106	64	23
26	0	16	2	103	59	21
27	2	15	1	97	53	17
28	1	12	0	94	50	14
29	1	11	1	88	43	9

Table 25. Transfer of airborne bacteria-carrying particles within a building, counts per 0.3 cubic feet of air sampled, samplers W and R.

Time Mins	Duty Room W	Small Ward R	Duty Room W	Large Ward R	Duty Room W	Large Ward R
0	8	36	1	0	6	0
1	14	36	2	0	3	0
2	30	298	3	0	4	1
3	45	401	5	0	4	0
4	57	452	2	0	2	0
5	65	396	7	1	2	0
6	48	368	4	0	0	1
7	47	358	8	0	1	2
8	42	343	10	2	1	1
9	40	316	11	0	0	1
10	37	304	17	1	2	0
11	35	289	13	1	3	0
12	31	272	16	0	7	0
13	30	256	13	0	4	0
14	27	244	8	0	3	1
15	28	218	10	2	2	1
16	26	202	11	0	0	0
17	25	189	7	1	0	0
18	21	168	7	0	2	2
19	20	159	4	0	0	0
20	19	138	8	0	4	0
21	17	145	7	1	2	0
22	18	141	6	2	0	1
23	15	136	3	0	2	0
24	14	123	4	0	2	1
25	12	117	5	1	1	1
26	11	113	2	0	1	0
27	13	108	2	0	0	0
28	10	100	1	0	1	0
29	9	97	3	0	0	1



Table 26. Plate-counts, per 0.1 ml., of bacterial suspension irradiated with ultraviolet light.

Time Secs	Colony counts - tube 6537/J					
	1	2	3	4	5	Mean
0	223	228	204	222	196	215
12	75	74	60	78	68	71
24	13	19	20	25	16	20
36	5	5	6	8	5	6
48	1	1	2	3	2	2
60	0	0	0	1	0	0

Time Secs	Colony counts - tube 3623/I					
	1	2	3	4	5	Mean
0	317	322	356	314	299	322
12	112	129	110	123	107	116
24	30	27	26	27	28	28
36	2	3	1	1	2	2
48	0	0	0	0	0	0
60	0	0	0	0	0	0

Time Secs	Colony counts - tube 6028/I					
	1	2	3	4	5	Mean
0	280	226	256	212	237	242
12	246	194	237	188	192	211
24	198	164	204	171	187	185
36	165	151	181	164	180	168
48	152	144	151	146	156	150
60	101	117	125	132	122	120

Table 27. Plate-counts, per 0.1 ml., of bacterial suspension irradiated with ultraviolet light.

Time Secs	Colony counts - tube V118/I					
	1	2	3	4	5	Mean
0	300	279	283	275	301	280
12	284	281	278	265	288	275
24	251	240	241	231	245	243
36	234	241	230	226	239	234
48	224	234	228	215	228	225
60	184	230	219	203	214	210

Time Secs	Colony counts - tube VY.1205/L					
	1	2	3	4	5	Mean
0	181	205	171	195	184	187
12	122	124	112	123	116	119
24	62	68	50	55	51	56
36	21	20	19	21	22	21
48	9	7	6	8	4	7
60	3	1	1	1	0	1

Time Secs	Colony counts - tube VY.7774/L					
	1	2	3	4	5	Mean
0	182	180	169	163	186	177
12	89	94	72	70	76	80
24	36	33	23	22	30	30
36	4	6	4	4	5	5
48	1	2	1	1	1	1
60	0	0	0	0	0	0

Table 28. Plate-counts, per 0.1 ul., of bacterial suspension irradiated with ultraviolet light.

Time Secs	Colony counts - tube VY.5855/L					
	1	2	3	4	5	Mean
0	174	169	189	185	171	172
12	84	82	94	104	87	90
24	68	48	55	63	59	59
36	46	33	40	41	42	41
48	30	31	30	33	32	31
60	21	19	20	24	16	20

Time Secs	Colony counts - tube VY.7290/P					
	1	2	3	4	5	Mean
0	202	234	186	172	191	197
12	134	109	119	113	130	119
24	57	67	71	70	63	66
36	24	27	18	23	21	23
48	4	5	5	4	6	5
60	0	2	1	1	0	1

Time Secs	Colony counts - tube VY.7950/K					
	1	2	3	4	5	Mean
0	190	178	180	236	214	200
12	193	164	170	210	180	178
24	118	84	84	132	104	104
36	74	52	40	70	63	61
48	34	33	26	28	24	30
60	10	14	12	8	4	10

Table 20. Plate-counts, per 0.1 ml., of bacterial suspension irradiated with ultraviolet light.

Time Secs	Colony counts - tube 7123/I					
	1	2	3	4	5	Mean
0	215	182	207	195	206	201
12	201	167	192	171	182	183
24	194	163	180	165	177	173
36	169	159	165	155	162	162
48	147	148	153	146	157	150
60	124	130	132	134	130	131

Time Secs	Colony counts - tube 6690/G					
	1	2	3	4	5	Mean
0	192	222	220	194	214	210
12	84	100	101	80	93	93
24	21	16	18	12	15	16
36	3	2	4	2	3	4
48	2	0	1	0	0	1
60	0	0	0	0	0	0

Time Secs	Colony counts - tube VV.7289/P					
	1	2	3	4	5	Mean
0	206	170	166	210	160	185
12	118	94	120	98	101	106
24	46	50	42	64	40	50
36	24	24	20	18	12	20
48	4	0	0	10	0	6
60	2	0	4	2	0	2

Table 30. Plate-counts, per 0.1 ml., of bacterial suspension irradiated with ultraviolet light.

Time Secs	Colony counts - tube 1462/K					
	1	2	3	4	5	Mean
0	220	235	211	220	221	221
12	105	97	100	110	109	104
24	30	31	20	36	35	30
36	4	4	2	3	2	3
48	0	0	1	0	0	0
60	0	0	0	0	0	0

Time Secs	Colony counts - tube 7127/I					
	1	2	3	4	5	Mean
0	228	215	223	231	221	224
12	191	187	190	193	190	190
24	165	161	160	166	161	163
36	140	138	136	133	134	139
48	123	116	120	117	118	119
60	103	99	101	98	98	99

Time Secs	Colony counts - tube N.2636					
	1	2	3	4	5	Mean
0	247	260	231	254	224	248
12	93	113	119	108	129	114
24	49	52	60	55	43	55
36	17	22	25	29	23	24
48	5	6	11	7	10	8
60	1	2	2	1	2	2

Table 31. Plate-counts, per 0.1 ml., of bacterial suspension irradiated with ultraviolet light.

Time Secs	Colony counts - tube VY.5370/L					
	1	2	3	4	5	Mean
0	212	187	197	201	192	198
12	118	97	96	102	99	104
24	58	49	44	47	50	49
36	38	39	30	37	36	36
48	24	21	17	20	23	21
60	17	14	13	16	16	15

Time Secs	Colony counts - tube 5367/X					
	1	2	3	4	5	Mean
0	202	190	191	196	182	192
12	174	173	161	156	152	164
24	109	105	117	109	112	110
36	86	89	93	90	91	90
48	77	75	61	66	79	69
60	51	59	59	63	57	57

Time Secs	Colony counts - tube VY.5362/L					
	1	2	3	4	5	Mean
0	249	240	221	243	242	240
12	128	146	165	136	169	149
24	87	81	59	82	63	74
36	27	30	35	26	24	28
48	8	7	7	8	9	8
60	6	5	3	6	4	5

Table 32. Plate-counts, per 0.1 ml., of bacterial suspension irradiated with ultraviolet light.

Time Secs	Colony counts - tube 0513/D					
	1	2	3	4	5	Mean
0	235	213	228	201	221	220
12	118	108	120	99	115	111
24	60	51	60	52	63	59
36	43	29	30	35	41	36
48	15	9	9	14	12	12
60	7	2	4	2	3	4

Time Secs	Colony counts - tube VV.7769/L					
	1	2	3	4	5	Mean
0	325	348	279	276	297	305
12	76	80	74	76	82	78
24	36	32	27	31	35	32
36	4	7	7	6	5	6
48	1	1	2	1	1	1
60	0	0	0	1	0	0

Table 33. Comparability of bacteriological and photometric methods for assessing ultraviolet irradiation.

Tube	Graph Slope 1 : x	Rank A	% Physical Efficiency	Rank B	A ± B
0537/J	0.09	5	69	6	1
3023/I	0.32	1	100	1	0
0028/I	0.70	18	21	12	0
7116/I	0.36	20	18	20	0
VY.1205/L	5.20	8	61	19	2
VY.7774/L	0.07	3	76	5	2
VY.5855/L	2.44	15	38	15	0
VX.7290/P	5.56	7	66	8	1
VY.7950/K	3.20	13	50	13	0
7123/I	0.56	19	20	19	0
0690/G	5.96	6	90	3	3
VY.7289/P	4.48	10	64	9	1
1452/K	6.41	4	98	2	2
7127/I	0.68	17	22	17	0
N.2838	4.76	9	63	7	2
VY.5870/L	2.68	14	48	14	0
5867/K	1.28	16	34	16	0
VY.5862/L	4.04	12	56	11	1
0513/D	4.08	11	52	12	1
VY.7700/L	7.45	2	86	4	2

Probability	Individual	cumulative
of equal ranking	0.45	0.45
of 1-rank difference	0.25	0.70
of 2-rank difference	0.25	0.95
of 3-rank difference	0.05	1.00



Table 34. Relationship of bacteriological and photometric methods for assessing ultraviolet irradiation.

Tube	Slope of bactericidal "decay curve"	Physical Efficiency
6037/J	-0.1022	69%
3623/I	-0.1436	100%
6028/I	-0.0110	21%
7116/I	-0.0054	18%
VY.1205/L	-0.0705	61%
VY.7774/L	-0.1078	76%
VY.6866/L	-0.0341	38%
VY.7290/P	-0.0759	66%
VY.7950/K	-0.0512	50%
7123/I	-0.0067	20%
6690/G	-0.1184	90%
VY.7289/P	-0.0710	64%
1452/K	-0.1191	96%
7127/I	-0.0134	22%
N.2836	-0.0819	68%
VY.6870/L	-0.0427	48%
6967/K	-0.0202	34%
VY.6862/L	-0.0703	56%
6613/D	-0.0677	52%
VY.7769/L	-0.1148	86%

**Table 35.** Counts of airborne bacteria-carrying particles settling per square foot of surface area per hour, in an operating theatre suite.

**Week 1:** Extract-only ventilation.  
19 operations - 1 emergency admissions' day

	Corridor	Vestibule	Theatre 1	Theatre 2
3pm - 8am	263	218	104	60
8am - 3pm	260	200	90	11
3pm - 8am	191	158	104	77
8am - 3pm	473	290	350	174
3pm - 8am	413	591	132	50
8am - 3pm	330	180	200	194
3pm - 8am	194	101	121	45
8am - 3pm	418	209	209	120

**Week 2:** Input-extract ventilation.  
18 operations - 1 emergency admissions' day

3pm - 8am	254	109	67	27
8am - 3pm	161	80	44	29
3pm - 8am	119	79	53	21
8am - 3pm	352	238	101	81
3pm - 8am	113	63	67	29
8am - 3pm	288	191	143	66
3pm - 8am	168	112	40	43
8am - 3pm	392	451	227	130

**Average counts for each period of day.**

3pm - 8am				
Week 1	372	220	229	128
Week 2	293	242	129	77
8am - 3pm				
Week 1	265	267	116	58
Week 2	194	91	60	30

Table 36. Counts of airborne bacteria-carrying particles settling per square foot of surface area per hour, in relation to a special chute system.

Time	Theatre D. U. R.				Ward 4 D. U. R.			
	A	B	C	D	A	B	C	D
1pm - 6pm	14	42	21	32	148	86	61	104
6pm - 8am	21	21	16	-	65	87	55	60
8am - 1pm	319	343	343	385	569	641	173	526
1pm - 6pm	604	420	402	498	314	241	73	92
6pm - 8am	53	57	40	46	205	179	96	78
8am - 1pm	442	233	310	432	739	1645	353	1050
1pm - 6pm	192	126	241	276	199	353	451	343
6pm - 8am	83	126	95	60	104	92	68	103
8am - 1pm	58	79	76	94	174	603	170	314
1pm - 6pm	48	75	31	132	105	120	47	39

Time	Ward 3 D. U. R.				Basement chute area			
	A	B	C	D	A	B	C	D
1pm - 6pm	144	129	74	105	94	199	105	35
6pm - 8am	72	75	36	44	18	30	18	13
8am - 1pm	130	203	95	154	70	67	46	67
1pm - 6pm	383	490	378	357	75	150	36	39
6pm - 8am	152	175	137	116	24	23	5	18
8am - 1pm	720	886	497	692	505	1045	388	514
1pm - 6pm	340	490	371	343	67	96	59	43
6pm - 8am	130	183	83	124	29	29	5	21
8am - 1pm	122	274	55	141	452	553	175	119
1pm - 6pm	100	96	63	67	319	294	256	200

Workmen active during period of sampling.

Table 37. Counts of airborne bacteria-carrying particles settling per square foot of surface area per hour, in relation to a simple chute system.

Time	Theatre D.U.R.		Basement	
	A	B	A	B
6pm - 8am	79	18	67	83
8am - 1pm	172	186	602	604
1pm - 6pm	504	422	248	346
6pm - 8am	1048	55	74	58
8am - 1pm	592	386	576	267
1pm - 6pm	189	263	106	99
6pm - 8am	101	53	88	99
8am - 1pm	310	195	356	403
1pm - 6pm	584	261	99	76

Time	Ward 8 D.U.R.		Ward 6 D.U.R.		Basement	
	A	B	A	B	A	B
6pm - 8am	359	463	223	298	86	147
8am - 1pm	1020	934	766	924	923	269
1pm - 6pm	538	488	486	1163	119	166
6pm - 8am	370	325	481	875	30	33
8am - 1pm	1430	838	1798	2578	252	278
1pm - 6pm	915	578	702	756	188	193
6pm - 8am	512	3209	449	1922	45	75
8am - 1pm	1193	2921	1144	1003	909	947
1pm - 6pm	455	1290	644	1049	167	122

Table 33. Average settle-plate counts relating to a special disposal-chute system.

		1pm - 6pm	6pm - 8am	8am - 1pm
Theatre D.U.R.	A	214 (4)	52 (0)	273 (0)
	B	106 (2)	68 (0)	220 (0)
	C	106	50	248
	D	234	53	204
Ward 4 D.U.R. (chute closed)	A	191 (0)	125 (0)	494 (0)
	B	202 (0)	110 (0)	903 (0)
	C	158	78	400
	D	144	60	630
Ward 3 D.U.R.	A	229 (1)	118 (1)	324 (6)
	B	300 (1)	147 (0)	454 (3)
	C	221	65	216
	D	193	93	331
Basement area	A	139	24	342
	B	186	29	557
	C	114	9	208
	D	81	14	233

Note: Figures in brackets indicate average number of times appropriate chute was used.

- A - sampling site at refuse chute.
- B - sampling site at linen chute.
- C - sampling site at window ledge.
- D - sampling site at washbasin, except basement which was at wall shelf.

Table 39. Average settle-plate counts relating to a simple disposal-chute system.

		1pm - 6pm	6pm - 8am	8am - 1pm
Theatre D.U.R.	A	448 (3)	409 (0)	358 (2)
	B	315	40	256
Basement area	A	151 (3)	76 (0)	578 (2)
	B	174	80	425

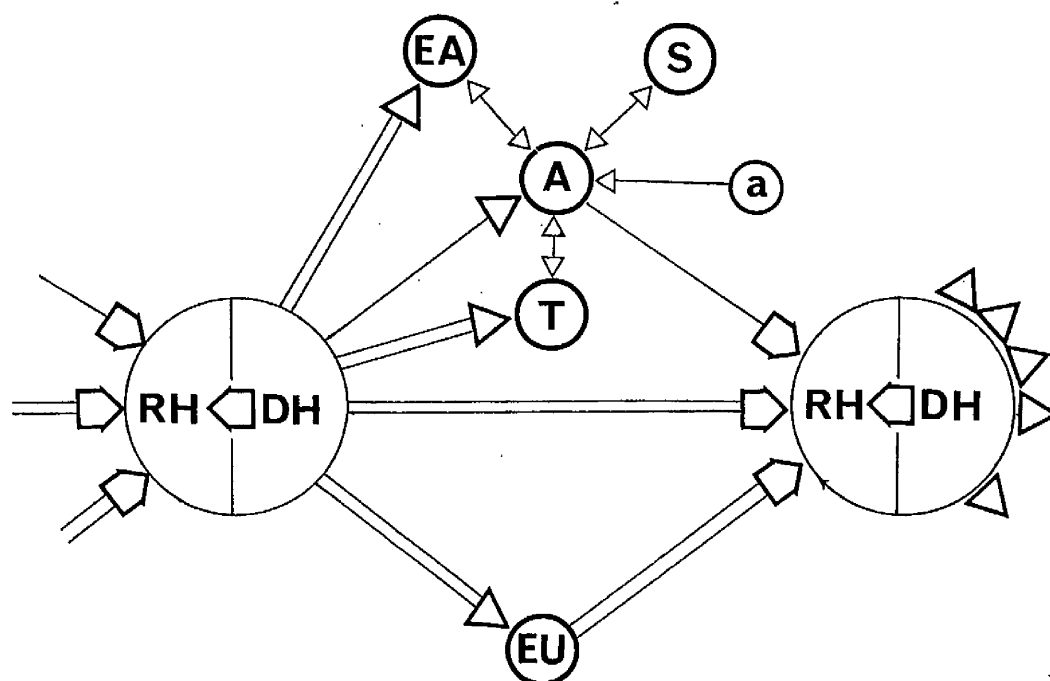
Ward 8 D.U.R.	A	638 (1)	414 (0)	1216 (2)
	B	785	1382	1564
Ward 6 D.U.R. (chute closed)	A	631 (0)	384 (0)	1206 (0)
	B	989	998	1502
Basement area	A	140 (2)	54 (1)	495 (4)
	B	160	87	498

Note: Figures in brackets indicate average number of times appropriate chute was used.

A - sampling site at linen chute.

B - sampling site on window ledge, except basement which was on wall shelf.

Figure 1.



Human Host

Environment

Human Host

Key: RH = Recipient host.

DH = Donor host.

A = Air inside hospital.

a = Air outside hospital.

EA = Equipment able to  
produce aerosols.EU = Equipment not able to  
produce aerosols.

T = Textiles.

S = Structures.

=⇒ = spread by contact.

=▷ = spread by dispersal.

◁ = endogenous spread.

Figure 2.

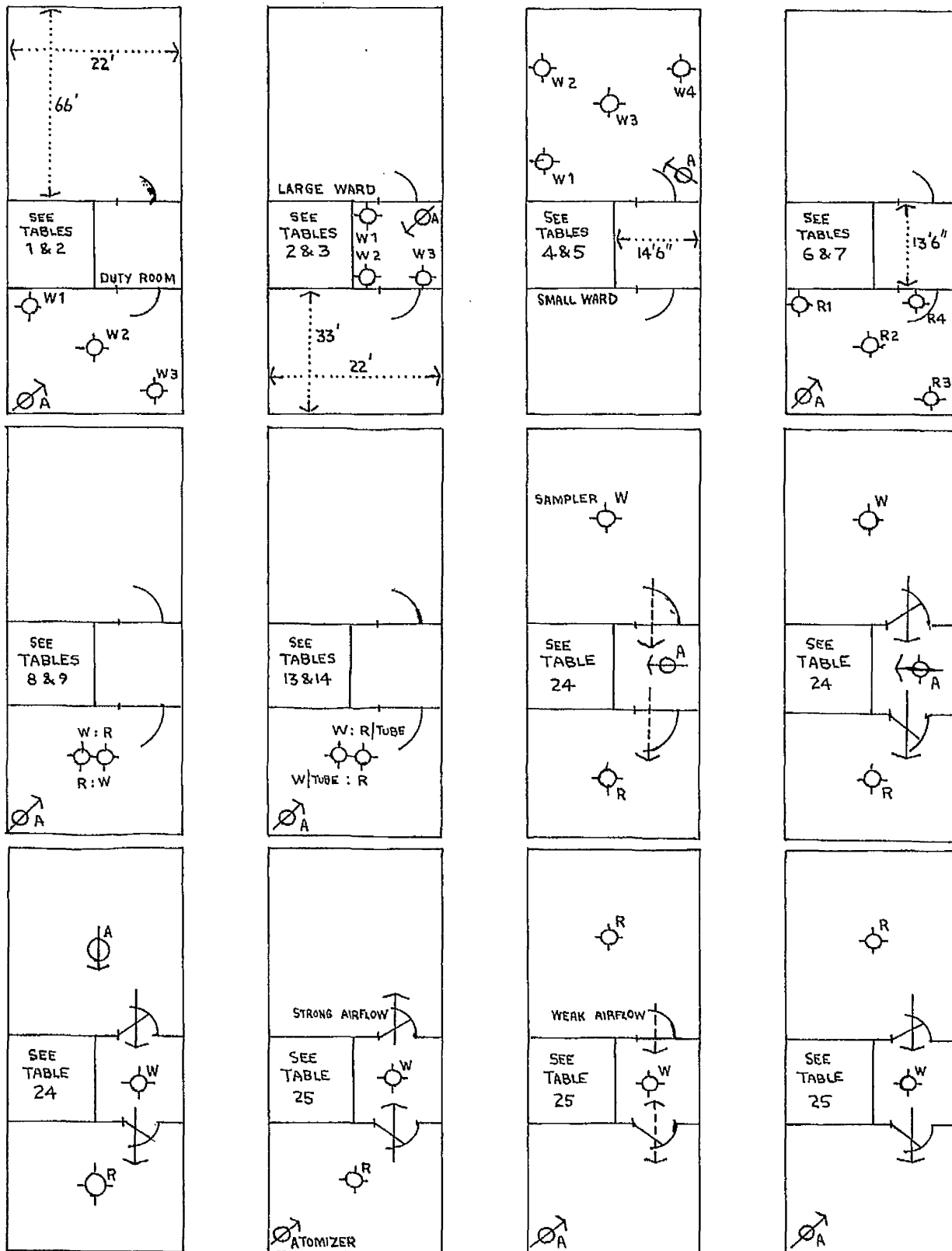




Figure 3.

Sampler W.  
Small Ward.

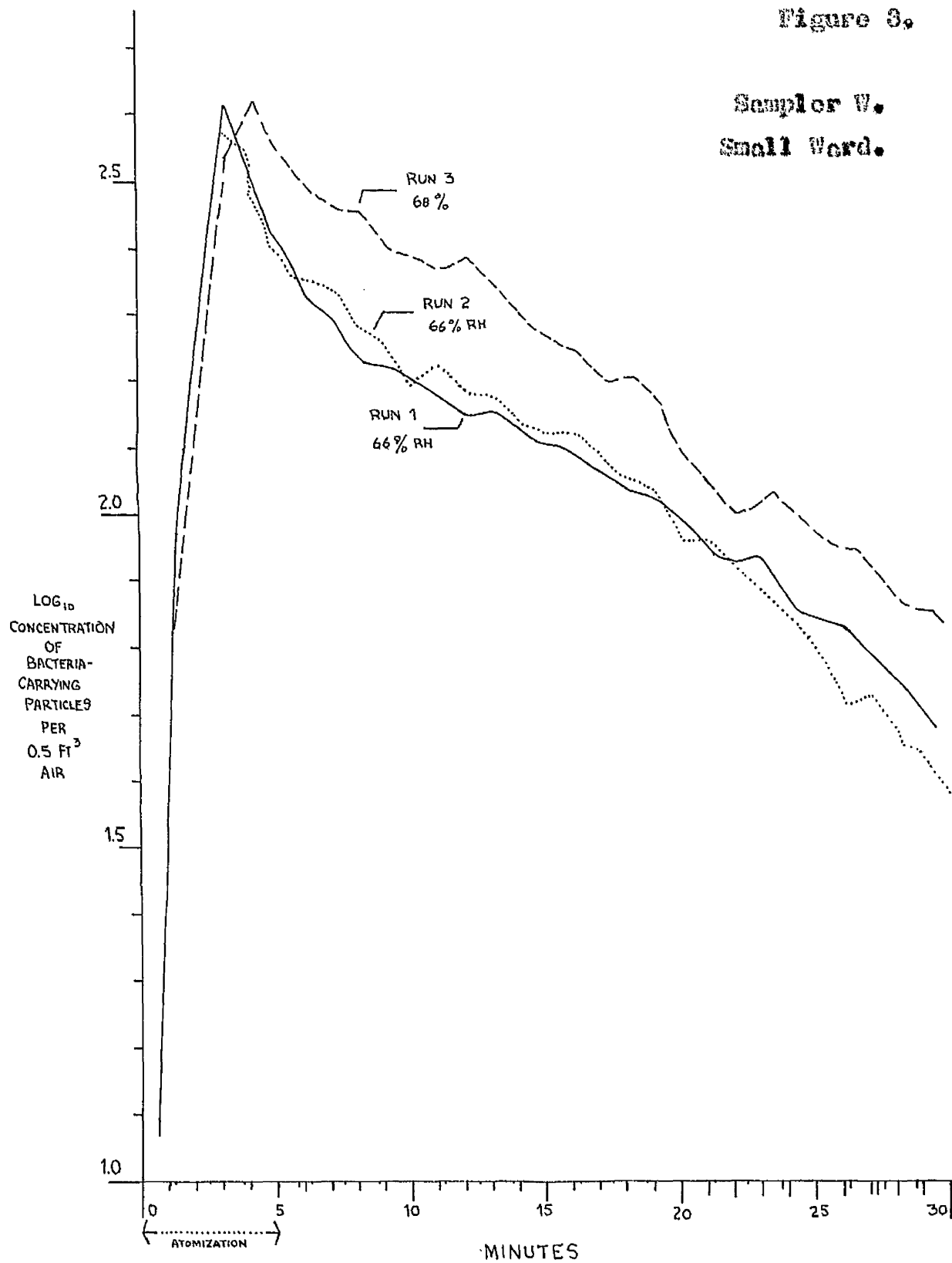
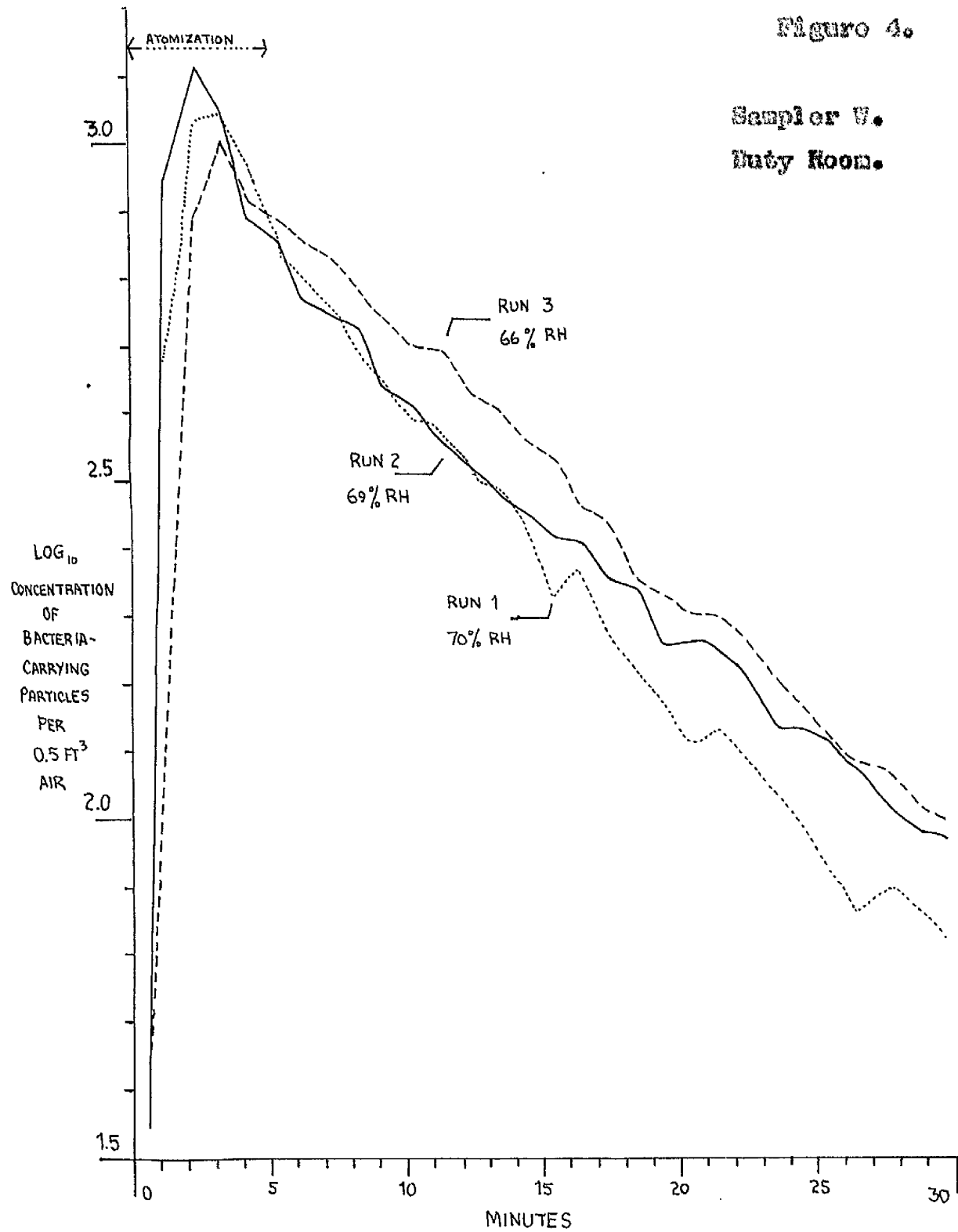


Figure 4.

Sampler W.  
Duty Room.



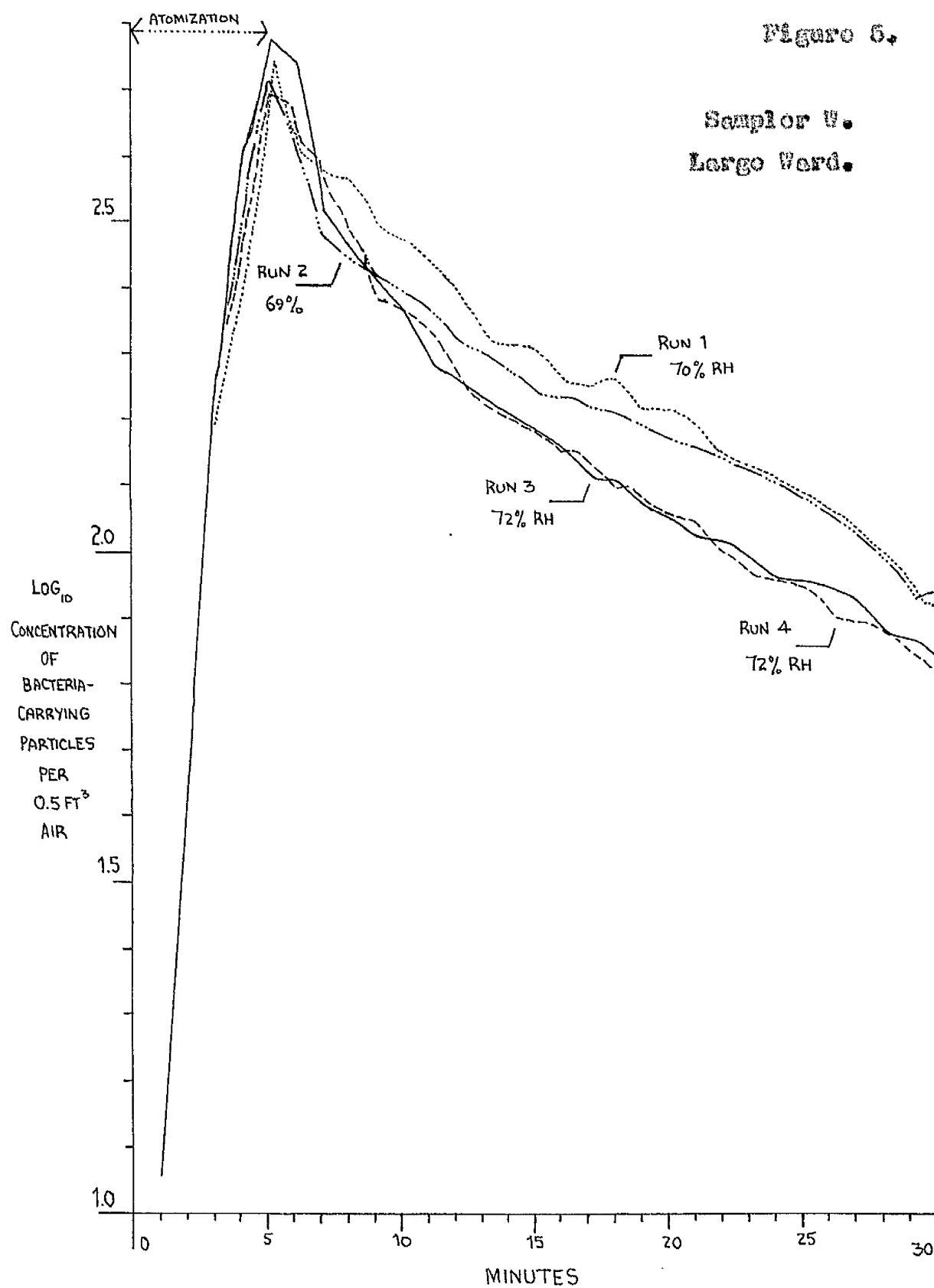


Figure 6.

Sampler R.  
Small Ward.

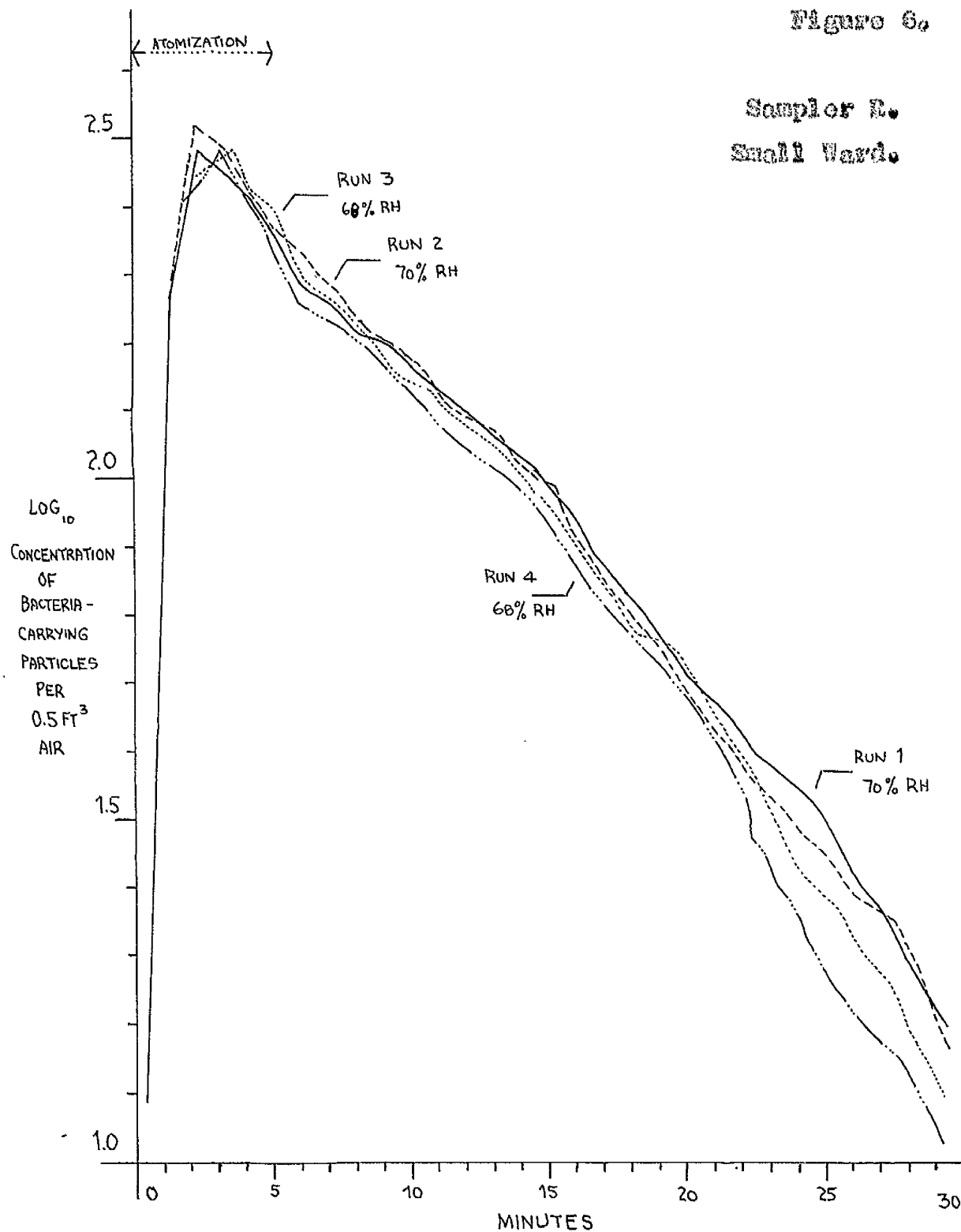


Figure 7.

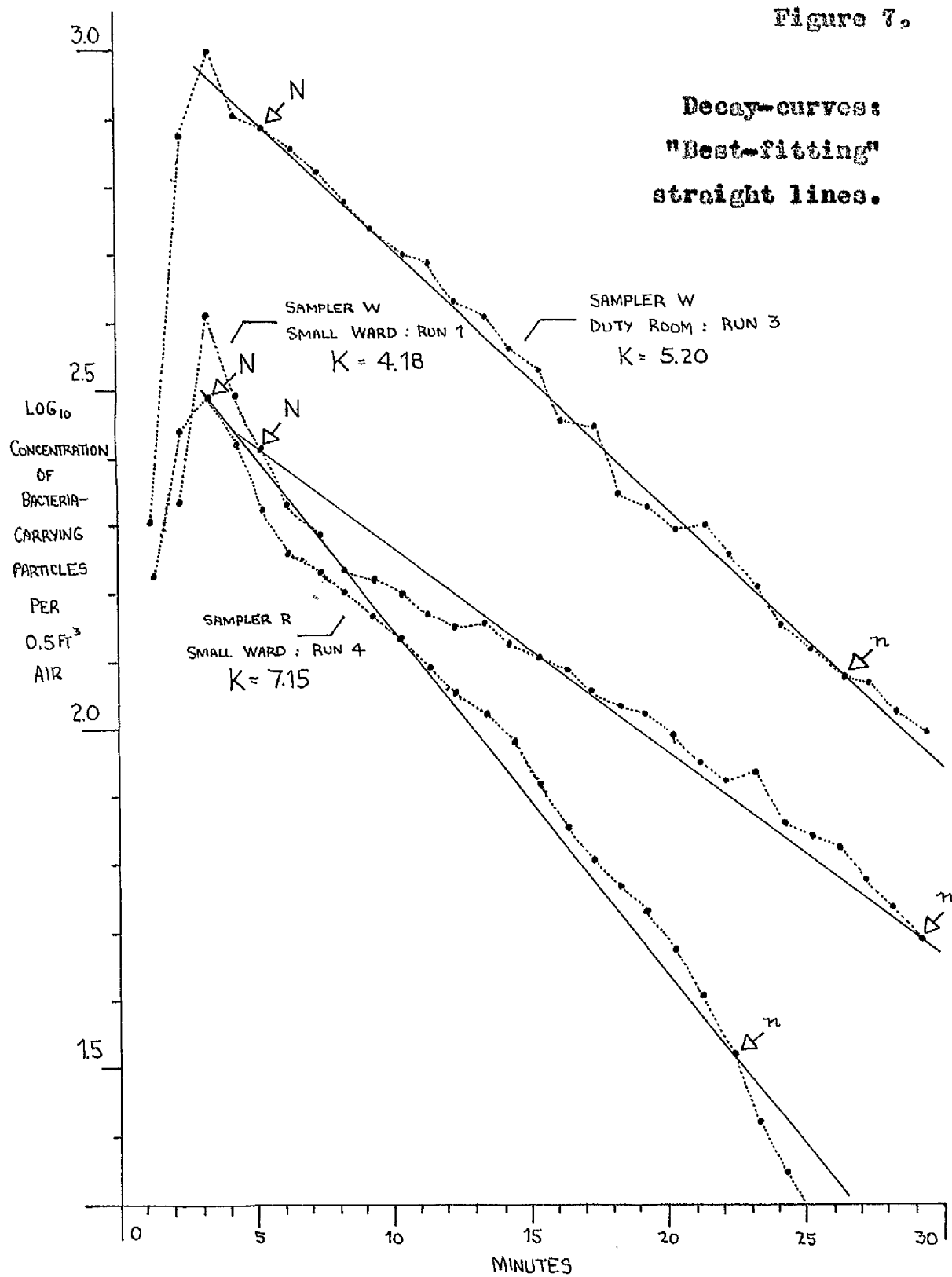


Figure 8.  
Colony counting  
"correction factors".

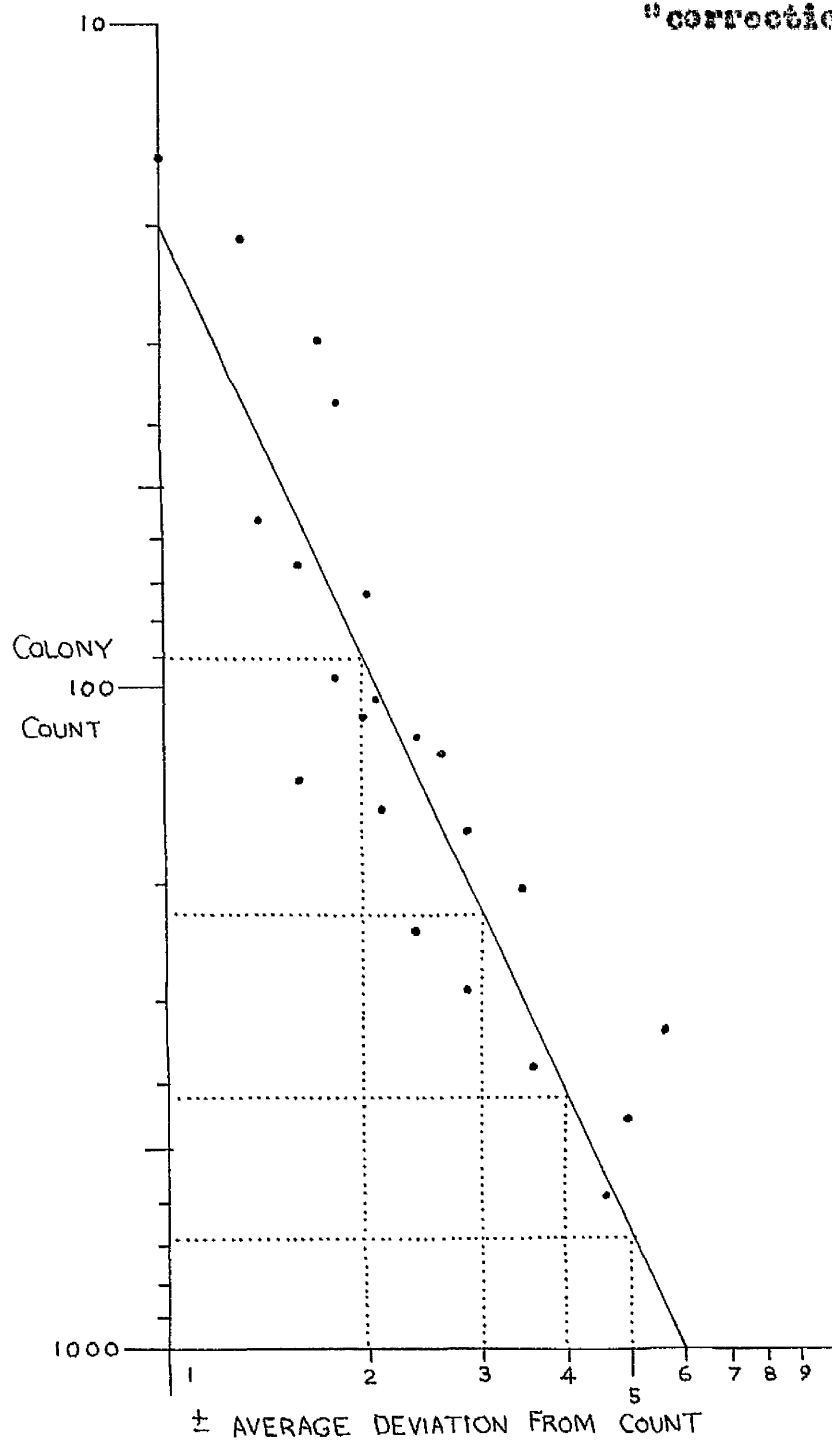


Figure 9.

Ultraviolet Irradiation,  
Bactericidal decay-curves.

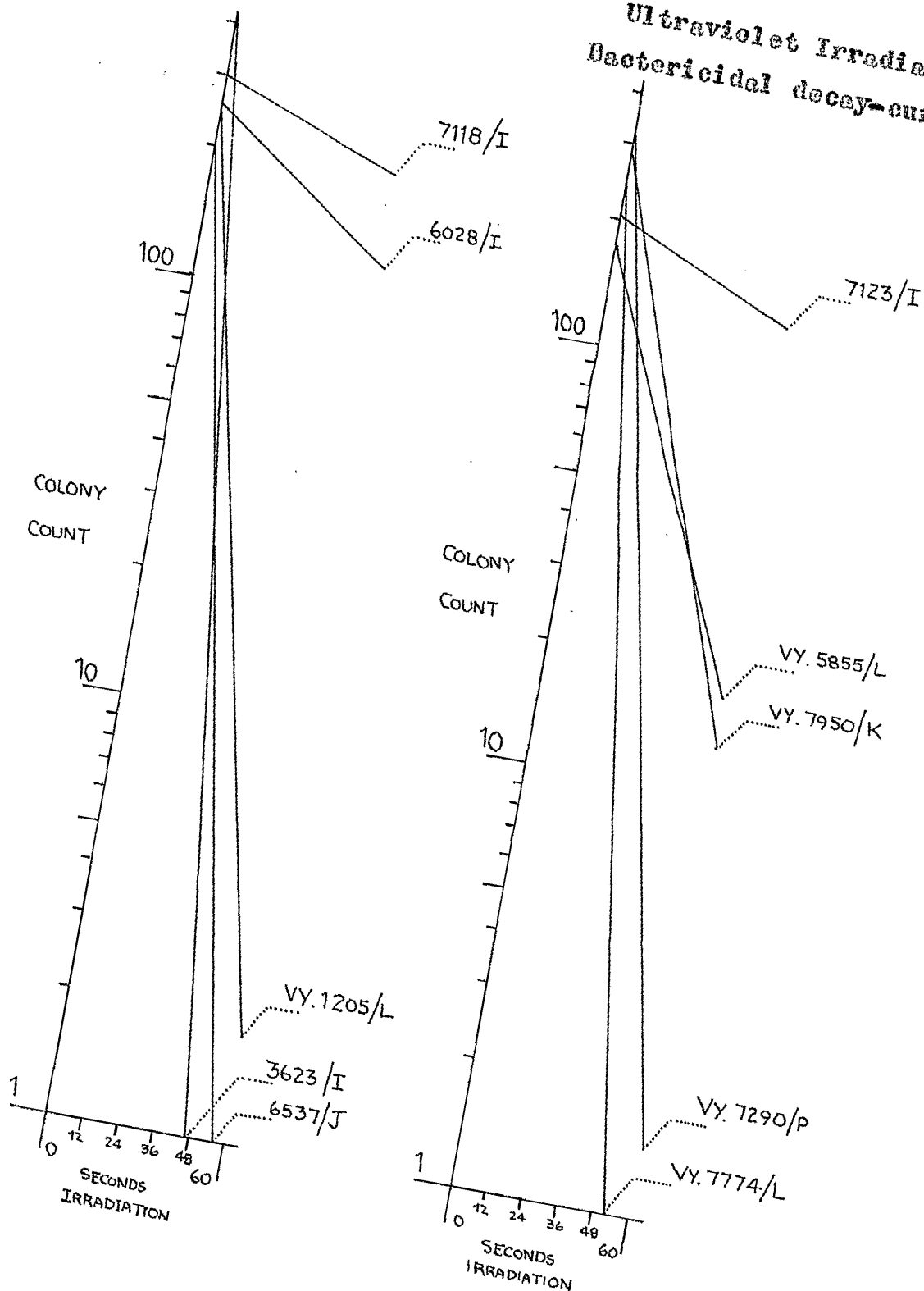


Figure 10.  
Ultraviolet Irradiation,  
Bactericidal decay-curves.

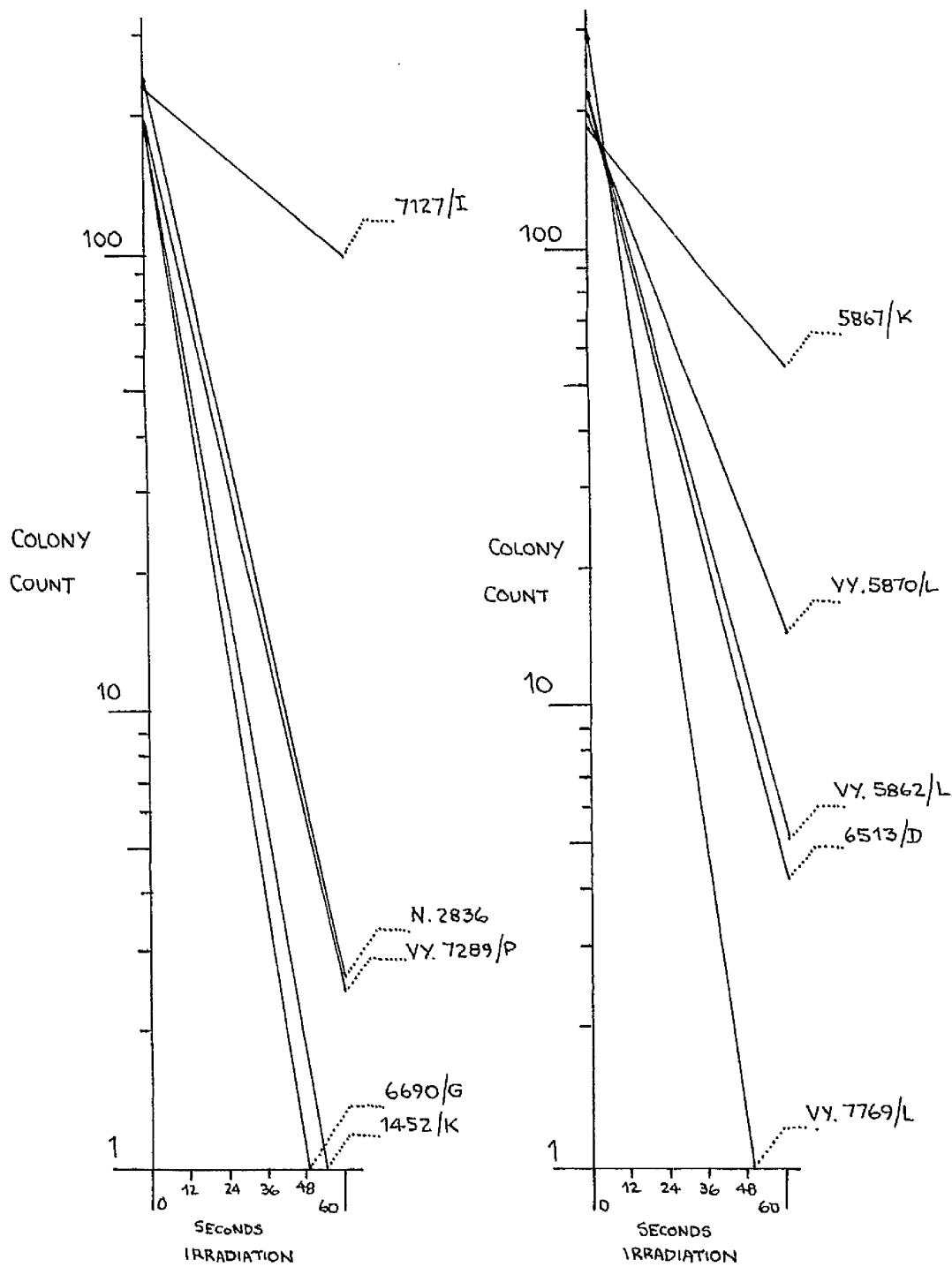




Figure 11.

Ultraviolet Irradiation,  
relationship of physical  
and bactericidal efficiencies.

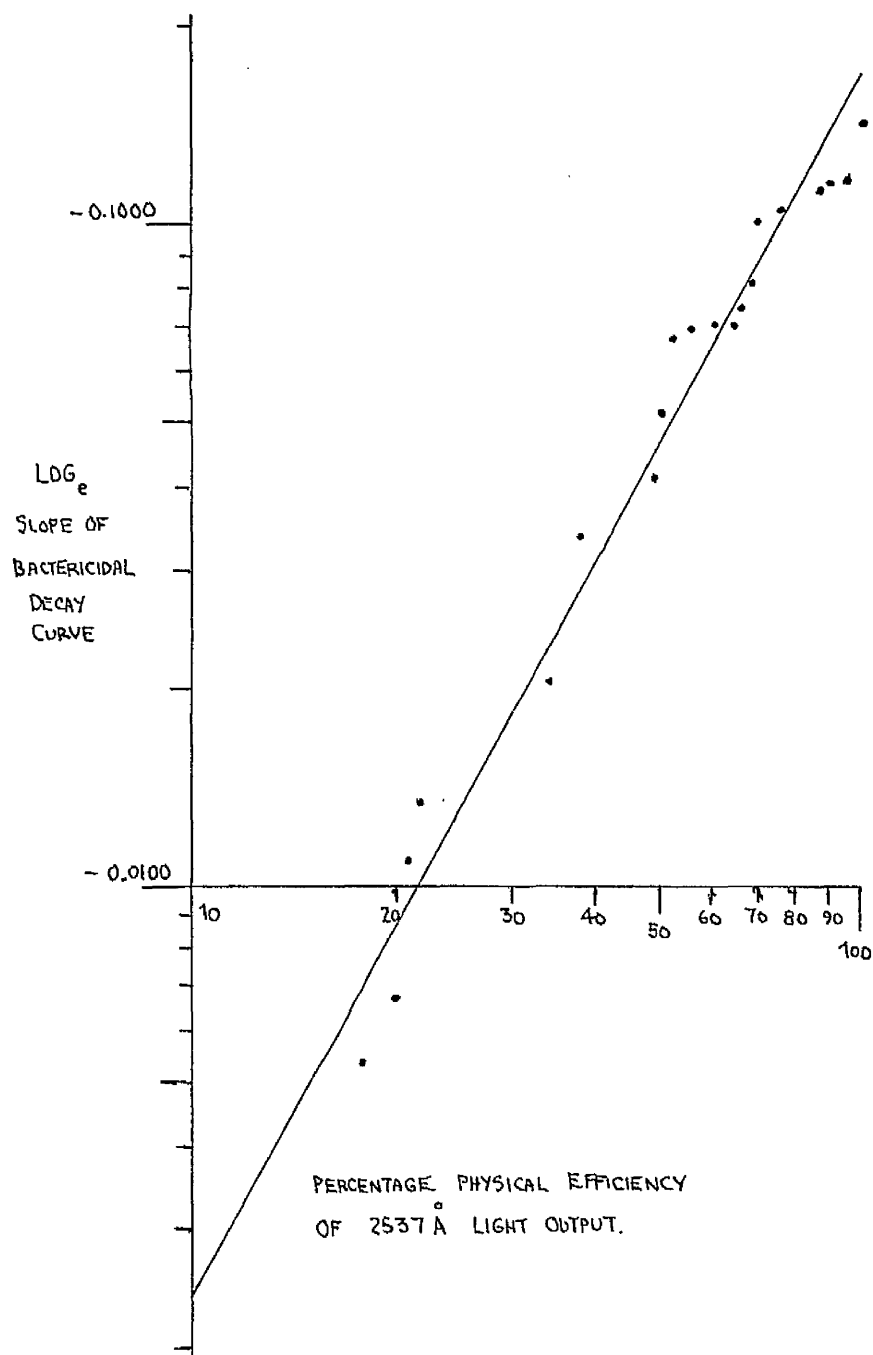


Figure 12.

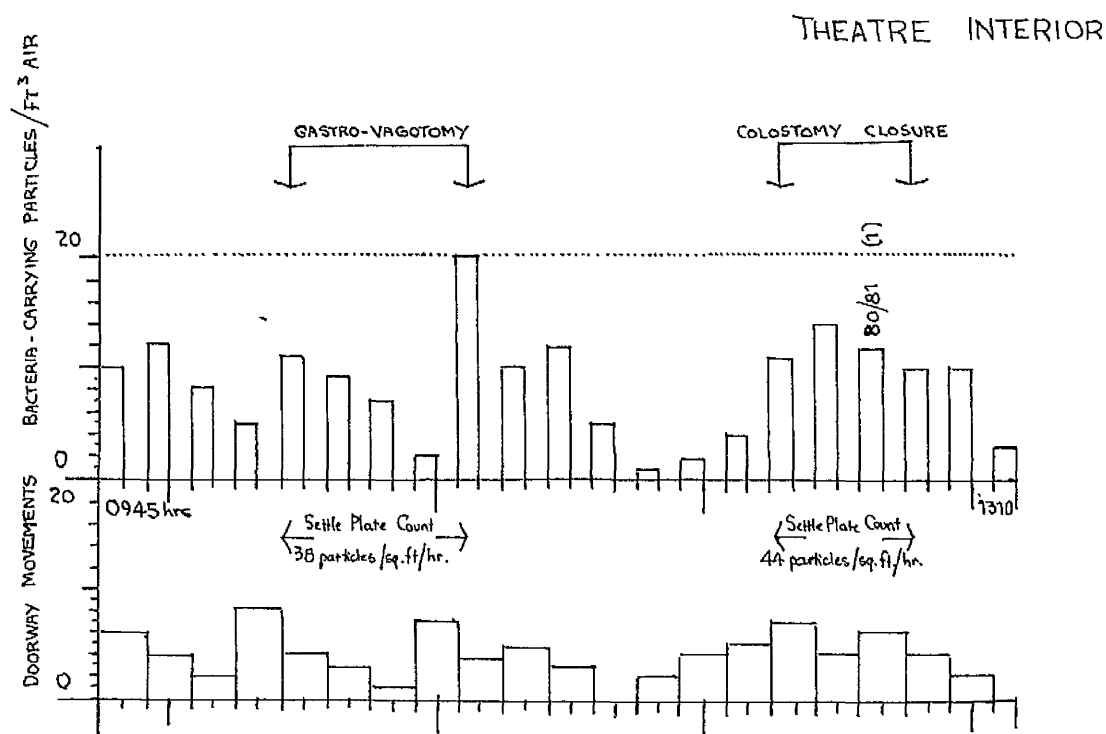
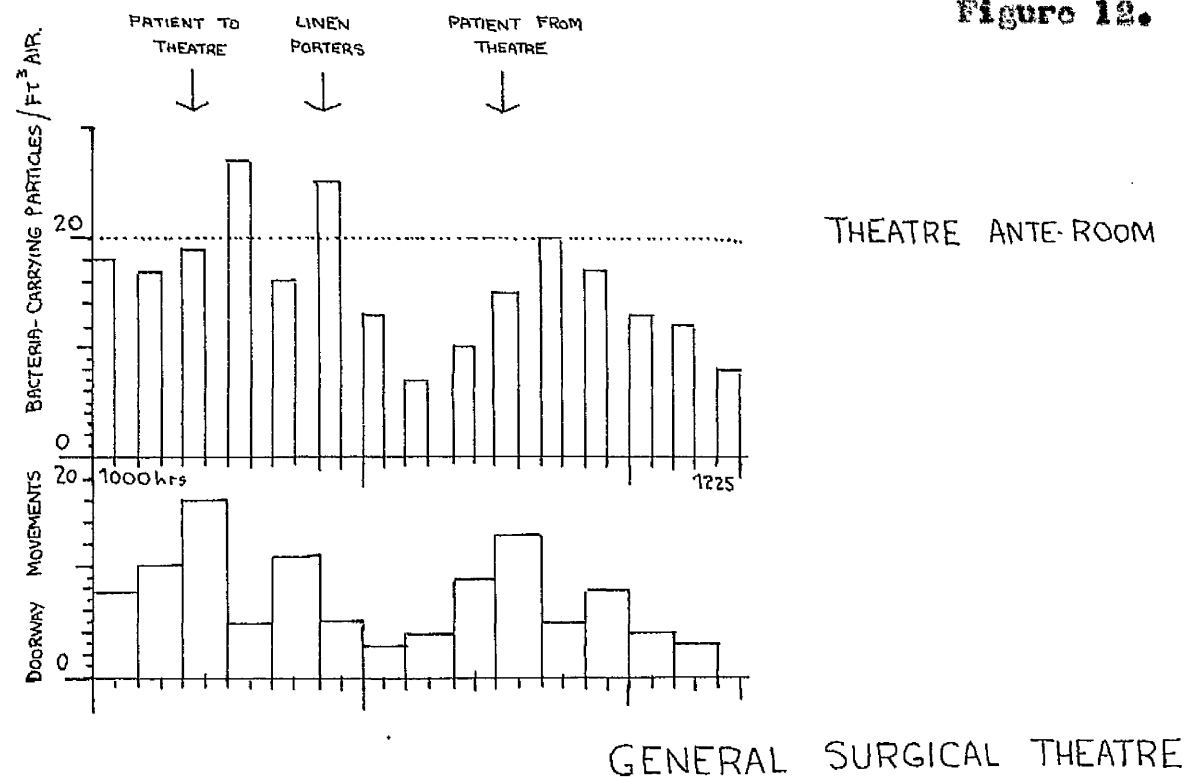
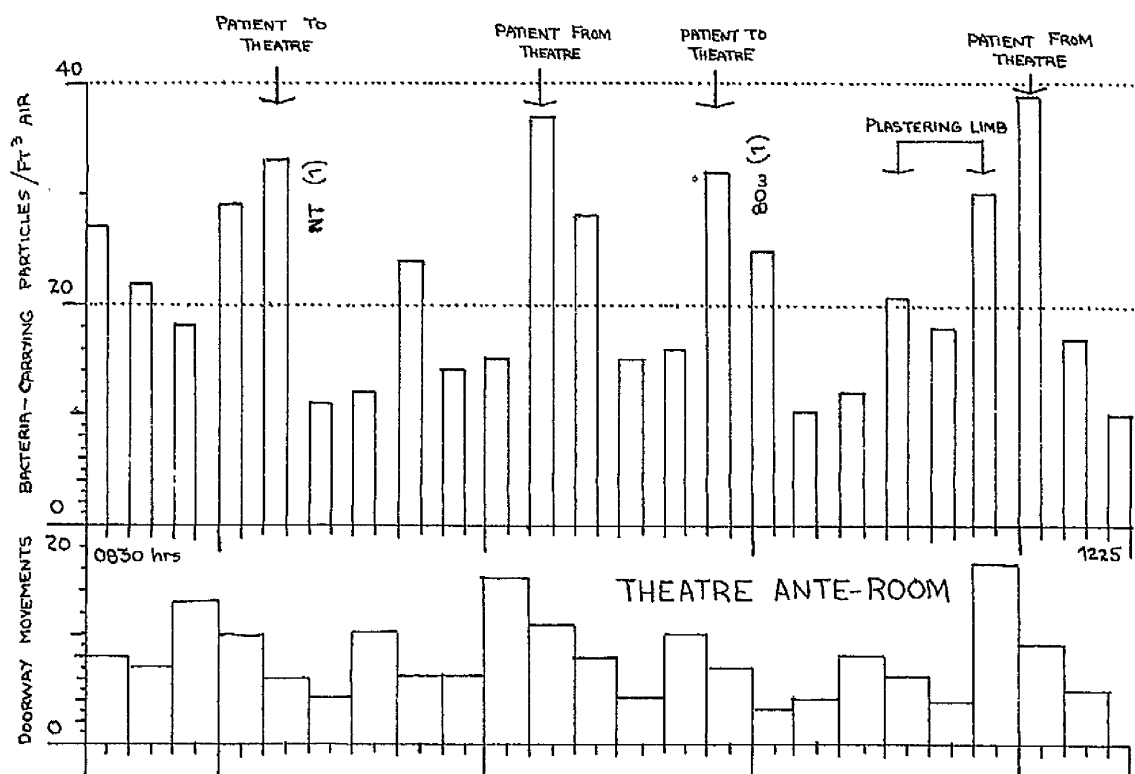
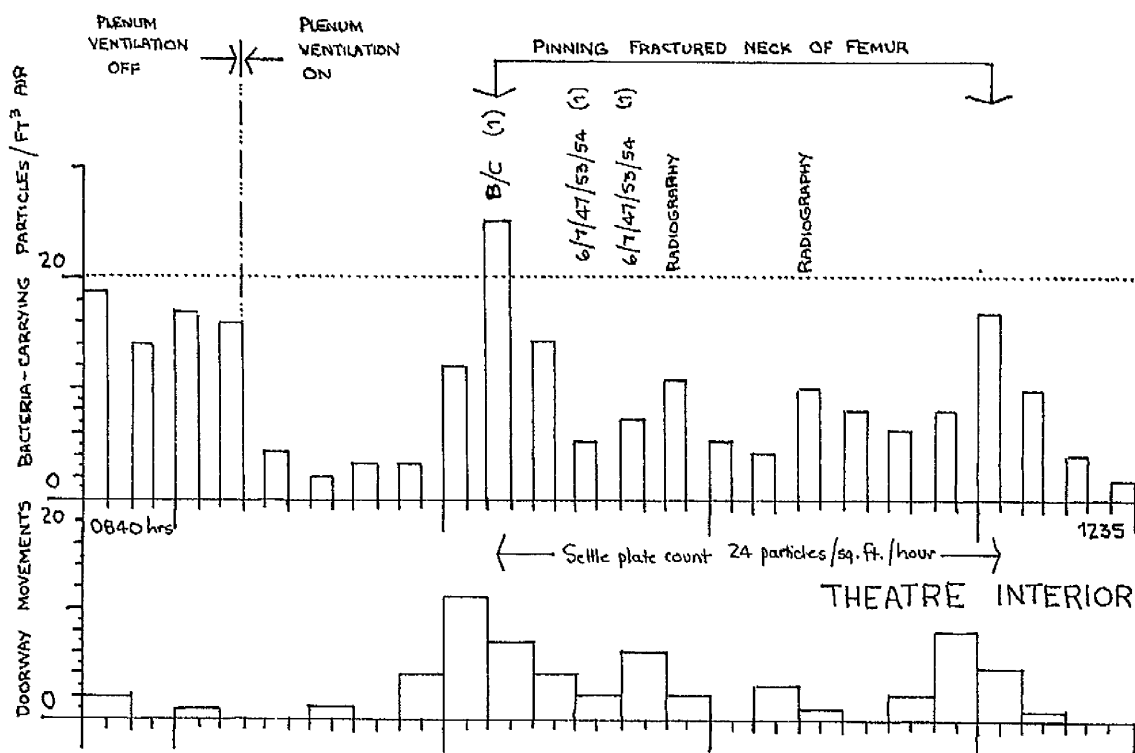


Figure 13.



## ORTHOPAEDIC THEATRE



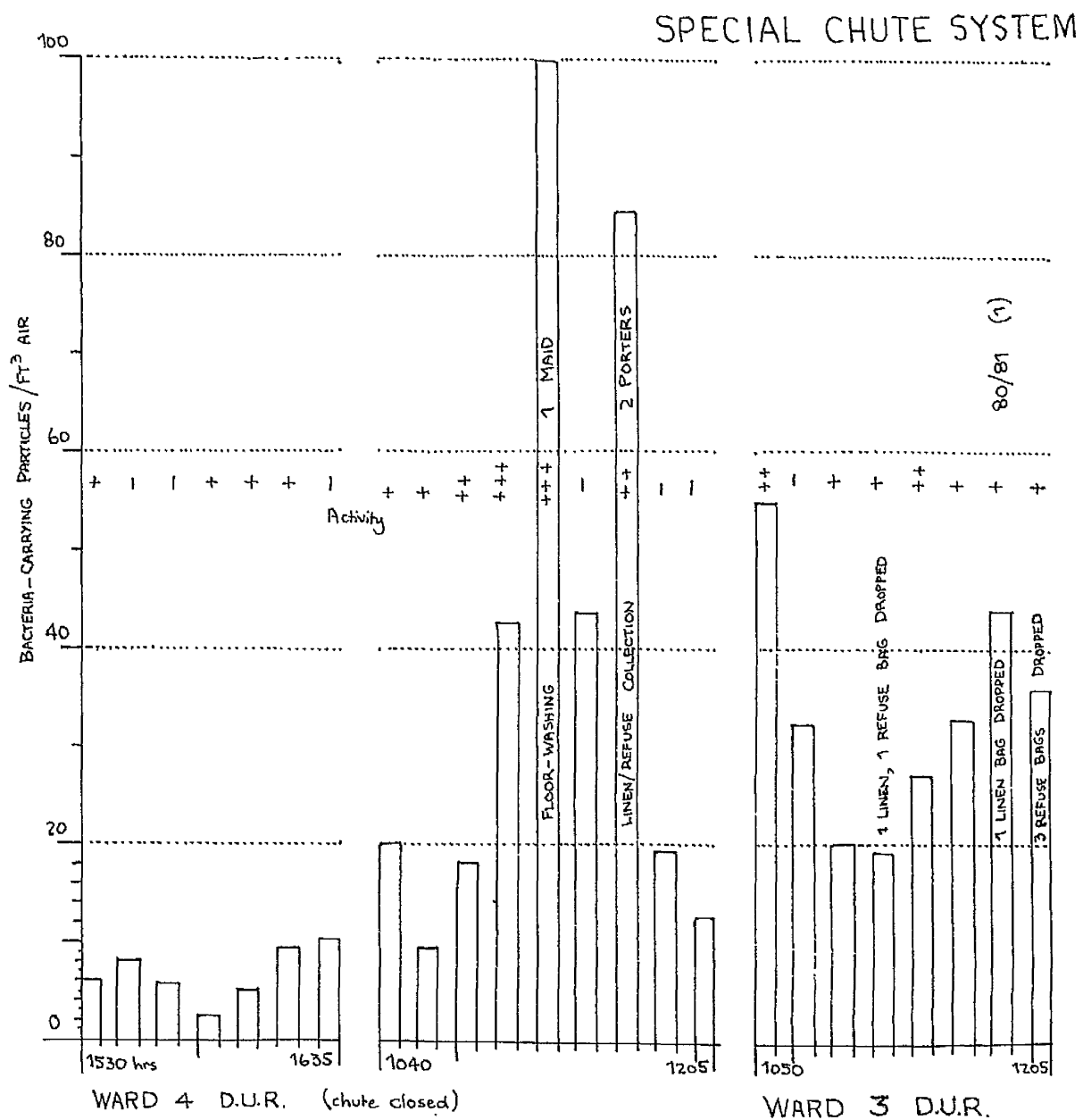
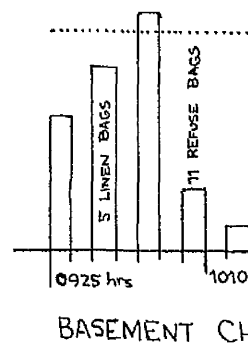


Figure 16.

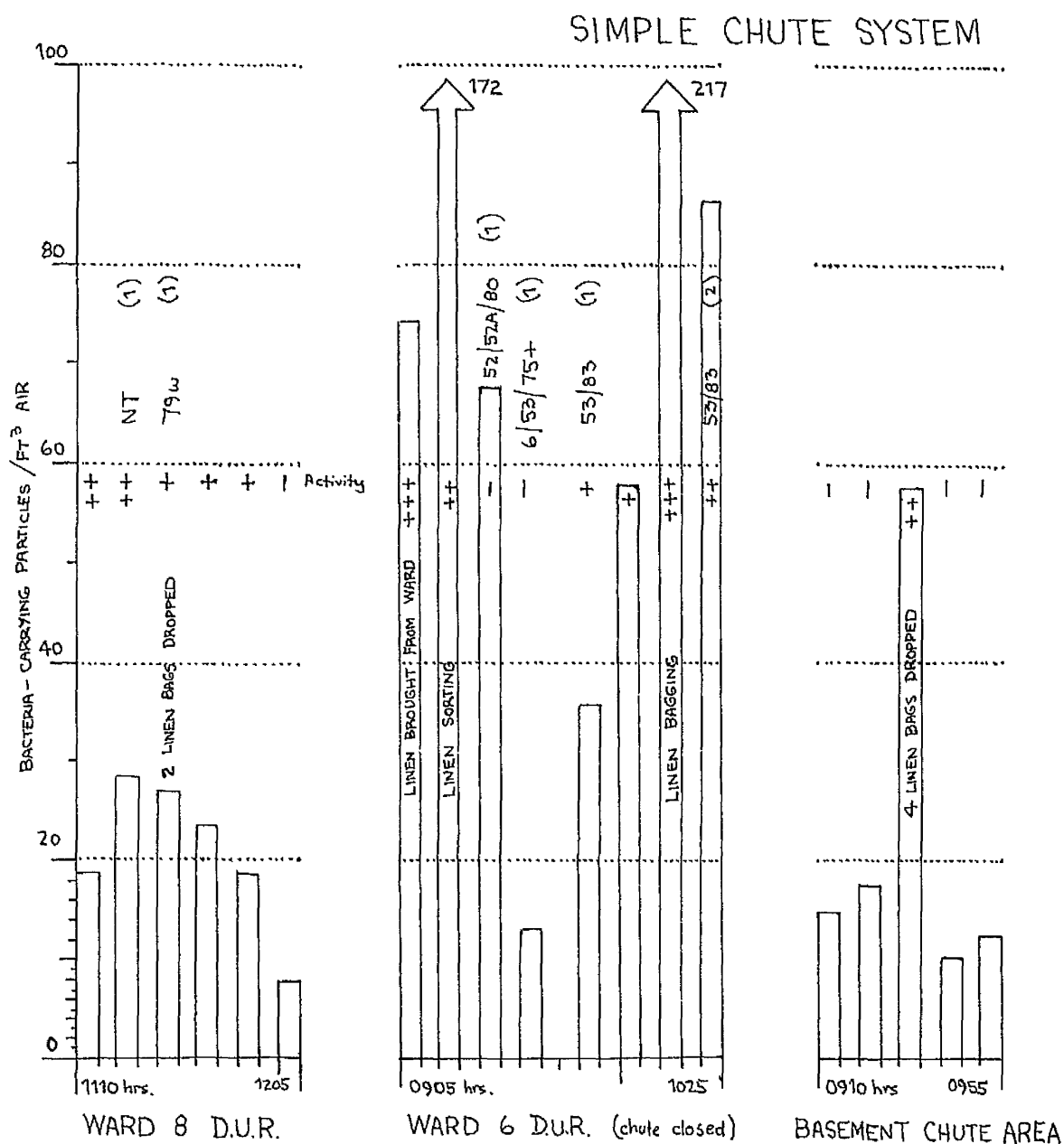
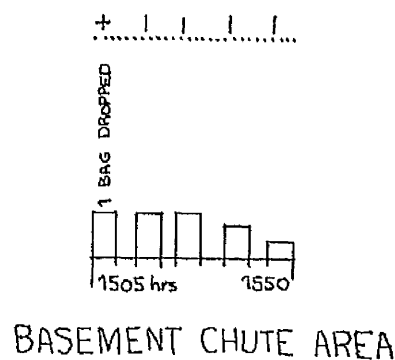
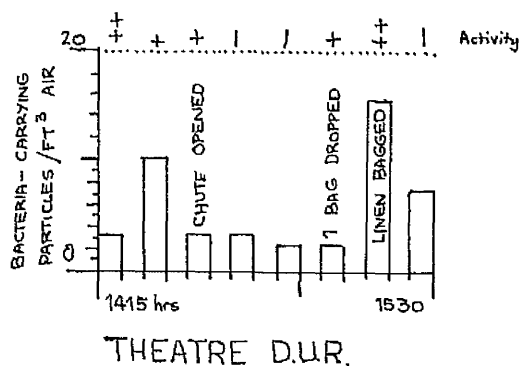
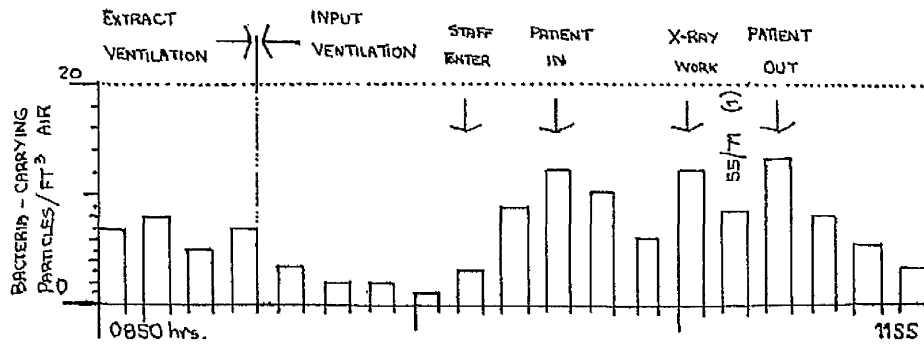
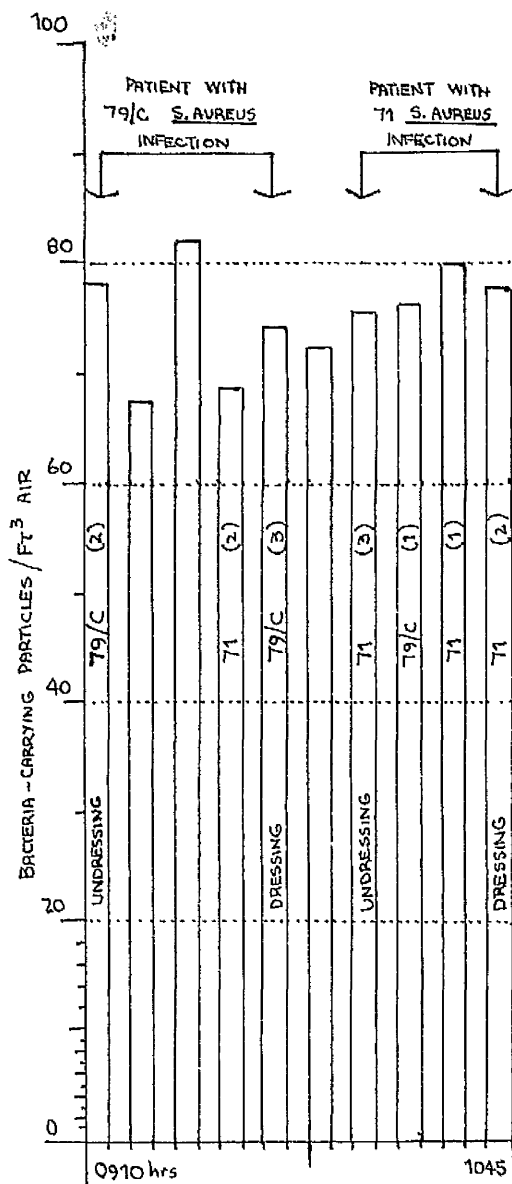


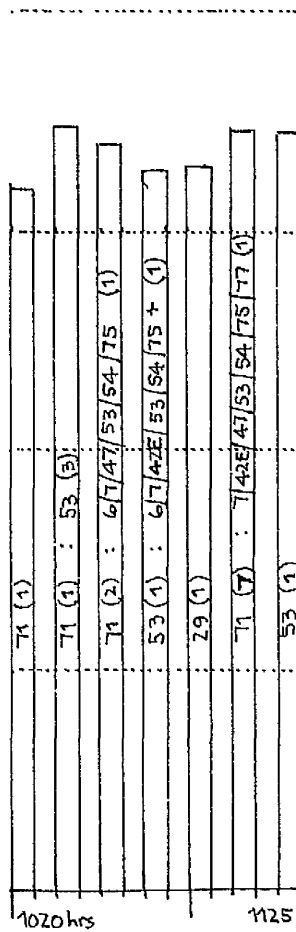
Figure 10.



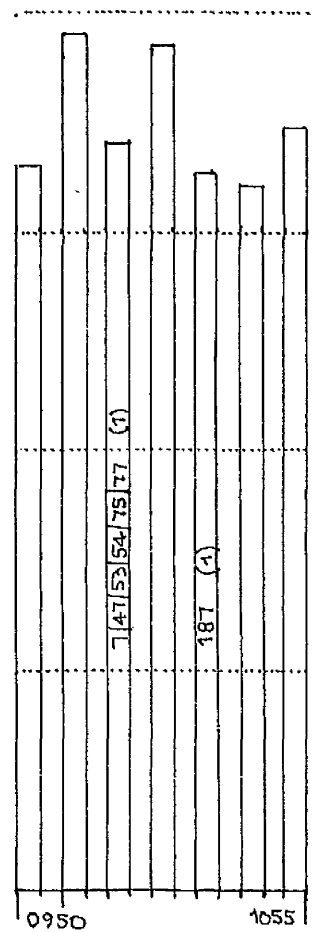
CARDIAC CATHETERISATION ROOM



TREATMENT ROOM



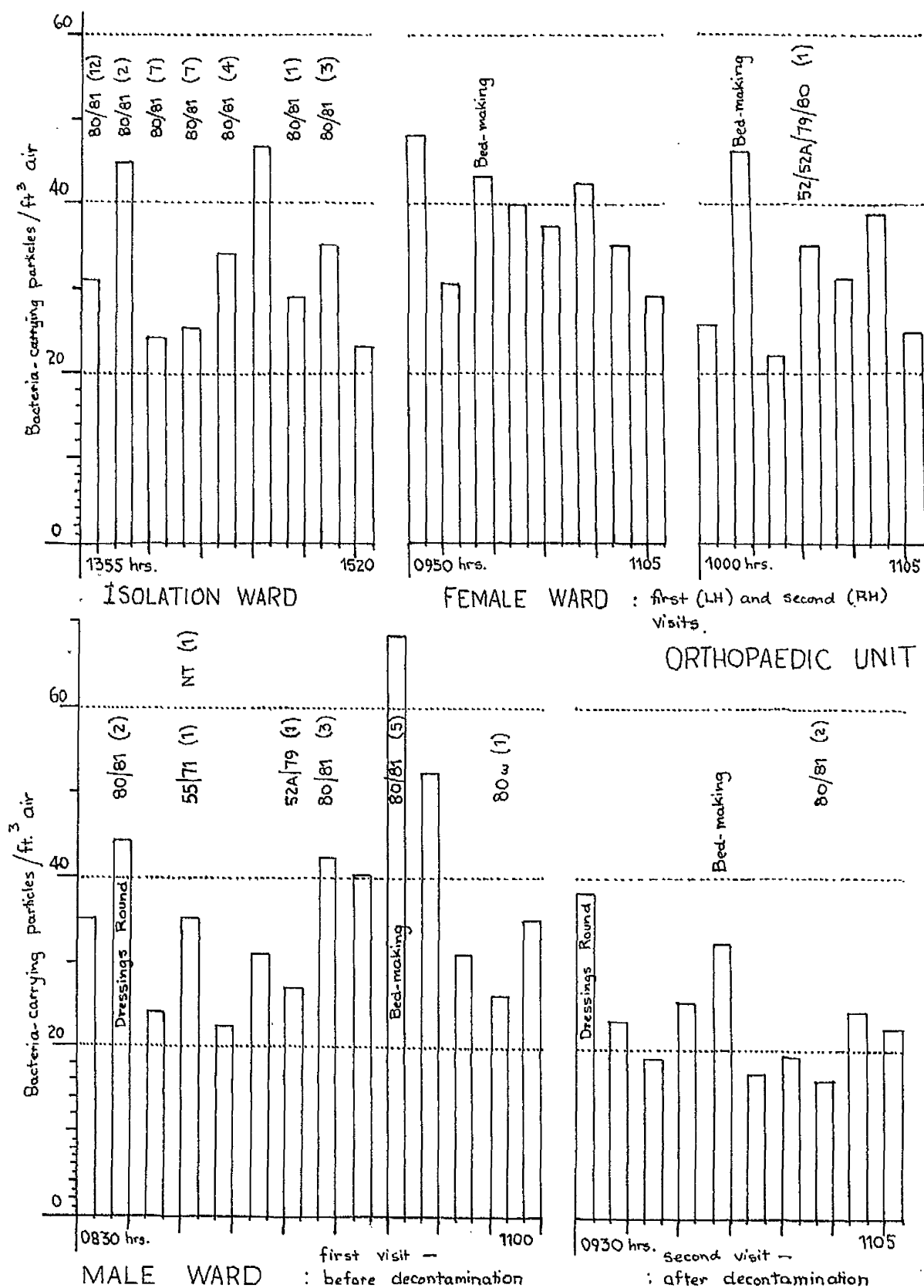
FEMALE WARD



MALE WARD

DERMATOLOGICAL UNIT

Figure 17.



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